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THE SURGICAL TREATMENT OF CYLINDROMA (ADENOID CYSTIC CARCINOMA) AND MUCO-EPIDERMOID TUMORS OF THE BRONCHUS

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THE CLASSIFICATION of primary cancer of the lung now in use at the Mayo Clinic includes a group of malignant tumors that originate in the mucous glands (Table I). The term "bronchial adenoma" is customarily used when discussing this group of tumors, which includes the carcinoid, the cylindroma, and the muco-epidermoid tumor. The term "adenoma," however, has a benign connotation which does not accurately describe the clinical behavior of many of these tumors.

The clinical features and the malignant potentialities of the carcinoid tumor have been the subject of many publications from this[1-3] and other institutions[4-8] and will not be discussed further. The present study is concerned primarily with the other two tumors of this group, the cylindroma (adenoid cystic carcinoma) and the muco-epidermoid tumor of the bronchus.

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HISTORICAL REVIEW

Heschl[9] in 1877 made the first report on cylindroma of the bronchus, and Müller[10] in 1882 was the first to describe what is now classified as a carcinoid tumor of the lung. In spite of the early recognition of these two types of tumor, their significance was not generally appreciated for many years. In the early 1930's, Kramer,[11] Geipel,[12] and Wessler and Rabin[13] distinguished a group of tumors called "adenomas" from the more common and more malignant bronchogenic carcinomas. In 1937, Hamperl[14] clearly distinguished the cylindroma from the carcinoid tumors within this "adenoma" group.

TABLE I. CLASSIFICATION OF PRIMARY CANCER OF THE LUNG

I. Tumors of bronchial epithelial origin
  A. Bronchogenic carcinoma
    1. Squamous cell carcinoma
    2. Adenocarcinoma
    3. Large cell carcinoma
    4. Small cell carcinoma
  B. Alveolar cell carcinoma (terminal bronchiolar)

II. Tumors of mucous gland origin ("bronchial adenoma")
  A. Carcinoid tumor
  B. Cylindroma (adenoid cystic carcinoma)
  C. Muco-epidermoid tumor

III. Tumors of mesenchymal origin
  A. Lymphosarcoma
  B. Fibrosarcoma

The term "cylindroma" was originated by Billroth[15] in 1859 to describe an orbital tumor. The term was subsequently used to describe the histologic appearance of certain tumors arising in the lacrimal and salivary glands and in the glandular structures of the breast and mucous membranes of the mouth, nose, paranasal sinuses, larynx, trachea, and bronchi.[16] The term "adenoid cystic carcinoma" is of more recent origin[17] and is used as a synonym for cylindroma as it accurately connotes the malignant nature and microscopic appearance of these tumors.

Muco-epidermoid tumors of the bronchus were first described by Liebow[5] and, as he has pointed out, they are identical histologically with the muco-epidermoid tumors originating in the salivary gland which Stewart, Foote, and Becker[18] described. In the past, these tumors have been provisionally classified by both Liebow[5] and us as cylindromas. Tumors of the muco-epidermoid type are included in the present discussion, not because they represent a histologic variant of cylindroma, but because this study of these lesions has led us to believe that they represent a distinct and separate clinical and pathologic entity which should not be confused with adenoid cystic carcinoma.

The chief histologic feature of adenoid cystic carcinoma (Fig. 1, *a*) is the arrangement of small, darkly staining cells in anastomosing solid or cystic cords. Between these collections of tumor cells are irregular areas of relatively acellular connective tissue which impart a "Swiss cheese" pattern.

The main feature of the muco-epidermoid tumors (Fig. 1, *b*) is the histologic pattern of an intimate admixture of well-differentiated mucous cells and

benign-appearing squamous elements, the cells of which may show intercellular bridges.

SCOPE OF PRESENT STUDY

In the present study we reviewed all the available clinical and pathologic material of all patients given a diagnosis of adenoma of the lower part of the respiratory tract at the Mayo Clinic in the 32-year period, 1927 through 1958. All tumors arising primarily in the trachea were excluded, but primary bronchial tumors showing tracheal involvement by direct extension were included. Also excluded from study were metastatic tumors to the lung.

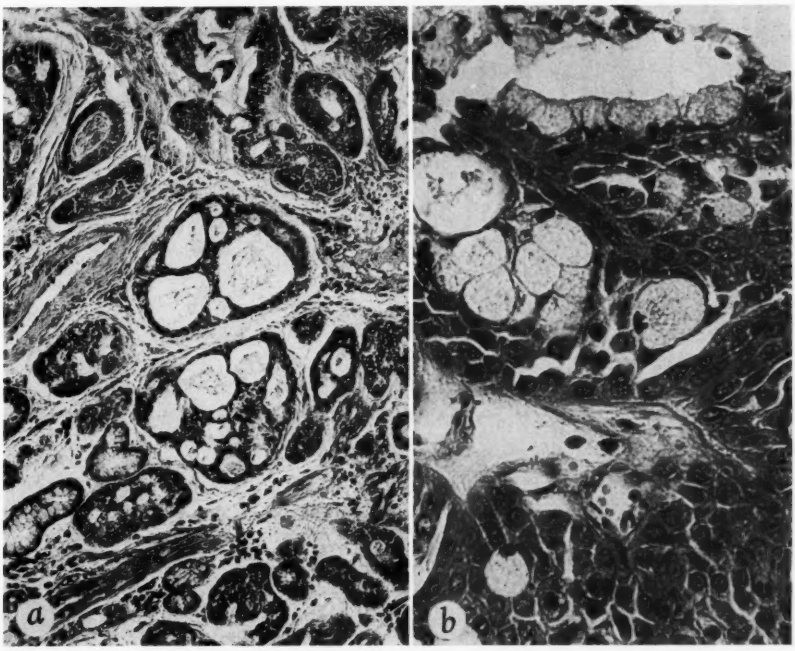

Fig. 1.—Sections showing the main histologic features. *a*, Adenoid cystic carcinoma (hematoxylin and eosin; ×130). *b*, Muco-epidermoid tumor (hematoxylin and eosin; ×400).

During this 32-year period, 162 so-called adenomas of the bronchus were encountered; 145 of these (89.5 per cent) were carcinoid tumors of the bronchus and will not be considered further; 13 (8.0 per cent) were adenoid cystic carcinomas of the bronchus, and 4 (2.5 per cent) were muco-epidermoid tumors (Table II).

TABLE II. TUMORS OF MUCOUS GLAND ORIGIN ("BRONCHIAL ADENOMA")

	CASES	PER CENT
Carcinoid tumors	145	89.5
Adenoid cystic carcinoma	13	8.0
Muco-epidermoid tumors	4	2.5
Total	162	100.0

TABLE III. CLINICAL AND PATHOLOGIC DATA ON PATIENTS

CASE	AGE (YR.) AND SEX	SYMPTOMS (YR.)	SITE IN BRONCHUS	LESION AT TIME OF TREATMENT				TREATMENT	RESULT
				EXTENSION		METASTASIS			
				MEDIASTINAL	TRACHEAL	NODAL	DISTANT		
				Cylindroma (Adenoid Cystic Carcinoma)					
1	42 F	1	R. main	+	+	?	?	Conservative	Died 2 yr. later; residual tumor
2	64 M	1	R. main	?	+	?	0	Conservative	Died 11 yr. later with metastases
3	36 M	1	L. main	0	+	0	0	Pneumonectomy (palliative)	Died 4 yr. later of metastasis (?)
4	50 M	6	R. lower lobe	+	+	+	0	Conservative; not resectable at exploration	Died 1 yr. later with metastasis
5	31 M	1½	L. main	?	+	?	?	Conservative	Died 9 mo. later; residual tumor
6	39 F	1½	L. main	0	0	+	0	Pneumonectomy	Alive 12¾ yr.; probable cure
7	48 M	15	R. lower lobe	0	0	0	0	Pneumonectomy	Died 10 mo. after operation of bronchopleural fistula; no residual tumor
8	60 F	¼	R. main	?	0	?	+	Conservative	Died 2 yr. later with metastasis
9	52 F	1	L. main	0	+	+	0	Conservative	Died 1 wk. after treatment; residual tumor
10	46 F	1½	R. main	+	+	+	0	Pneumonectomy (palliative)	Alive 4½ yr.; residual tumor
11	31 F	1	R. lower lobe	0	0	0	0	Right middle and lower lobectomy elsewhere	Dead 8 yr. later of widespread metastasis
12	45 F	4	L. lower lobe	+	+	+	0	Pneumonectomy (palliative)	Alive 2½ yr.; residual tumor
13	45 M	1	R. main	0	+	+	0	Pneumonectomy (palliative)	Alive 1 yr.; residual tumor
				Muco-Epidermoid Tumors					
14	65 M	6	R. main	0	0	0	0	Conservative	Died 2 wk. later; tumor present
15	44 M	⅙	R. upper lobe	0	0	0	0	Conservative	Died 4 yr. later; tumor present
16	47 F	3	L. main	0	0	0	0	Pneumonectomy	Alive 10 yr.; probable cure
17	46 F	2	R. main	0	0	0	0	Pneumonectomy	Alive 8 yr.; probable cure

CLINICAL REVIEW OF CASES

Age and Sex.—The ages of the 13 patients with adenoid cystic carcinoma ranged from 31 to 65 years, with an average age of 45 years at the time of diagnosis. Six patients were men and 7 were women (Table III).

The 4 patients with muco-epidermoid tumors ranged in age from 44 to 65 years, averaging 50½ years. They too showed no sex predilection. All patients in this study were of the Caucasian race.

Symptoms.—The average duration of symptoms of patients with adenoid cystic carcinoma was 3 years at the time of diagnosis. Although cough or wheeze was the most common symptom of long duration, usually some more recent and urgent symptom caused the patient to seek medical attention (Table IV). Only 1 patient (Case 8) was devoid of respiratory symptoms. Her chief complaint was referable to a metastatic adenoid cystic carcinoma involving the optic chiasm and the pituitary. The primary lesion in the lung was found incidental to routine thoracic roentgenography.

The average duration of symptoms of patients with muco-epidermoid tumors was about 3 years at the time of diagnosis. All had primary pulmonary complaints, and none had evidence of metastasis (Table IV).

TABLE IV. PRIMARY SYMPTOMS

	ADENOID CYSTIC CARCINOMA (13 CASES)	MUCO-EPIDERMOID TUMORS (4 CASES)
Cough	12	4
Hemoptysis	8	3
Dyspnea	7	3
Pain in thorax	7	1
Wheeze	9	4
Chills and fever	6	4
Loss of weight	8	1
Symptoms referable to central nervous system	1	0

The symptoms of both groups of patients were mainly those of bronchial irritation, or bronchial obstruction and pneumonitis. It was apparent that patients with muco-epidermoid tumors were more often acutely ill with suppurative complications than were those with adenoid cystic carcinoma.

Interestingly, none of the patients of either group had any clubbing of digits, although evidence of chronic pulmonary suppuration was apparent. None complained of arthritis, hoarseness, or symptoms of vasomotor instability suggestive of a functioning tumor.

Roentgenographic Appearance.—Roentgenographic examination of the thorax was of considerable importance in the study of these patients. The original roentgenograms of approximately half of the patients were available for review: those for 7 of the patients with adenoid cystic carcinoma and for 2 of those with muco-epidermoid tumors of the bronchus. The descriptive reports of the roentgenologists only were available for the remaining 8 patients.

The thoracic roentgenograms of 2 of the 13 patients with adenoid cystic carcinoma of the bronchus showed no abnormality at the time of diagnosis. The roentgenograms of 7 of the remaining 11 patients showed lobar collapse (Fig. 2, *a*), and those of 4, massive collapse of one entire lung similar to that seen in Fig. 2, *b*. In 3 of the patients with evidence of lobar collapse, a mass was also demonstrable roentgenographically.

The thoracic roentgenograms of all 4 of the patients with muco-epidermoid tumors showed abnormalities, and all manifested collapse of one entire lung (Fig. 2, *b*).

None of the thoracic roentgenograms taken at the time of diagnosis showed signs of cavitation, pleural fluid, obstructive emphysema, or involvement of bone. Furthermore, none of the roentgenograms studied presented signs sufficiently distinctive that one type of tumor could be differentiated from the other or from some other processes causing bronchial obstruction.

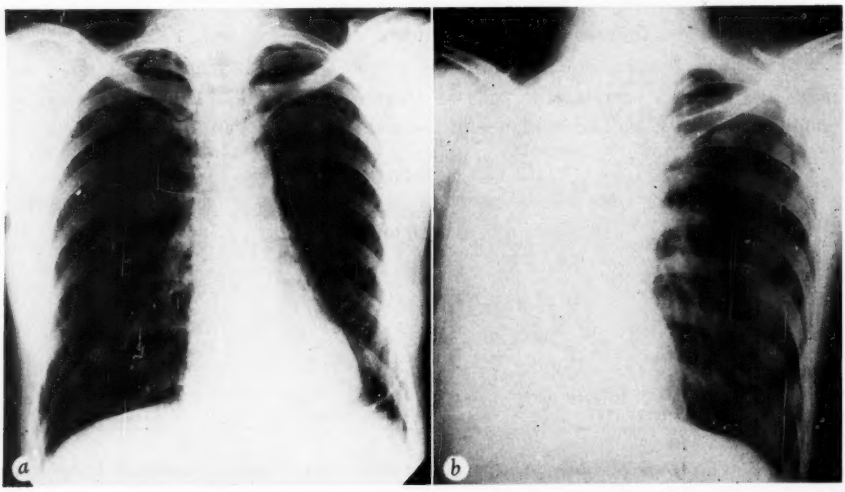

Fig. 2.—Thoracic roentgenograms. *a*, Patient (Case 12) with adenoid cystic carcinoma showing collapse of left lower lobe of the lung. *b*, Patient (Case 14) with a muco-epidermoid tumor of the right main bronchus producing massive collapse of one entire lung.

Bronchoscopic Appearance.—Bronchoscopic examination had been performed on all of the patients in this study. All of the tumors arose from either a primary or secondary bronchus, and all but one could be seen at the time of bronchoscopic examination. Endoscopic biopsy was the chief means of establishing the histologic diagnosis, and adequate specimens were obtained for diagnosis from all tumors that could be visualized. Thus, the diagnosis was made for all 13 patients with adenoid cystic carcinoma by this means and for 3 of the 4 patients with muco-epidermoid tumors. The only patient in whom the tumor could be neither seen nor biopsied was a patient with an extrabronchial muco-epidermoid tumor which produced bronchial obstruction by external compression (Case 15).

Each type of tumor had a distinctive endoscopic appearance. The adenoid cystic carcinomas of the bronchus presented as polypoid infiltrative tumors that partially or completely obstructed a bronchus. Often the tumor was covered with necrotic material and bled easily on manipulation. Grossly, they had the general appearance of bronchogenic carcinoma. Four of the 13 patients with adenoid cystic carcinoma showed signs of gross carinal or tracheal involvement at the time of the initial endoscopic examination.

The usual bronchoscopic description of muco-epidermoid tumors was that of a large polypoid endobronchial mass that completely obstructed a primary bronchus, presented a smooth clean surface, and did not readily bleed on manipulation. None of these tumors presented signs of infiltration or tracheal involvement. The single tumor that could not be visualized had caused bronchial obstruction that appeared endoscopically to be due to external compression.

Evidence of bronchial infection was present in all of the patients at the time of initial examination, but it was most marked in those with muco-epidermoid tumors. All 4 of these patients were described as having copious amounts of pus flowing from the region distal to the tumor, whereas only 1 of the patients with adenoid cystic carcinoma had such massive drainage at the time of initial bronchoscopic examination.

Cytologic Studies.—Cytologic studies were carried out on the sputum or bronchial secretion or both of 4 patients. Two of these were patients with adenoid cystic carcinoma, and malignant cells were identified in the bronchial smears or secretions from each. In 1 of these 2 patients, clumps of cells were identified which were suggestive of adenoid cystic carcinoma.

Cytologic studies on the 2 patients with muco-epidermoid tumors of the bronchus did not reveal any abnormal cells.

STUDY OF THE GROSS AND MICROSCOPIC PATHOLOGY

Information regarding the gross appearance and extent of spread of these tumors which will be presented in this portion of the paper was obtained from a review of gross surgical specimens available, the descriptive operative reports of surgeons in these cases, and from a review of pathologic data and material obtained at the time of necropsy. Although microscopic sections were available in all 17 cases in this study, and were reviewed, additional sections were obtained for microscopic study when the gross specimens were available in order to document the characteristics of these tumors further. We shall present here only the details concerning these additional microscopic studies. Findings with regard to extension of the lesion at time of treatment are given in Table III. They were derived from all sources then available and are more inclusive than those we shall now present.

Adenoid Cystic Carcinoma.—In 7 of the 13 cases of adenoid cystic carcinoma, the gross specimen of lung containing the primary tumor was available for review. Six were surgical specimens and one was obtained at postmortem examination.

The distribution and area of gross involvement of the tumor found in these seven specimens of lung is schematically illustrated in Fig. 3, *a*. All of these tumors involved major bronchi, and none presented as a peripheral pulmonary lesion. The typical gross appearance of the adenoid cystic carcinomas was that of an infiltrating malignant lesion (Fig. 4). The endobronchial surface of these

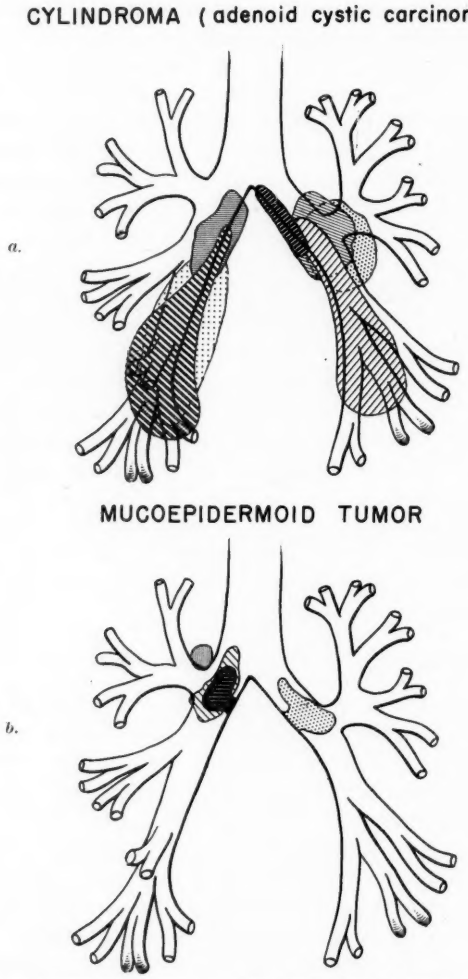

Fig. 3.—*a*, Outline of tumor involvement in seven specimens of lungs from patients with adenoid cystic carcinoma. *b*, Location and gross form of four muco-epidermoid tumors.

tumors was nodular and polypoid and involved the bronchial wall in a tubular fashion. Over the ulcerating endobronchial surface, the tumors were covered with necrotic material. The walls of major bronchi were thickened and indurated. Two of the seven specimens showed gross involvement of the tracheobronchial junction. Pulmonary parenchymal involvement was not unusual,

and when it occurred, small bronchi appeared as cylinders filled with inspissated secretions passing through a dense mass of tumor. Occasionally peribronchial lymph nodes were included in the tumor and showed gross involvement. In one of the seven specimens, tumor was evident on gross inspection in a large pulmonary vein. It was apparent from the operative

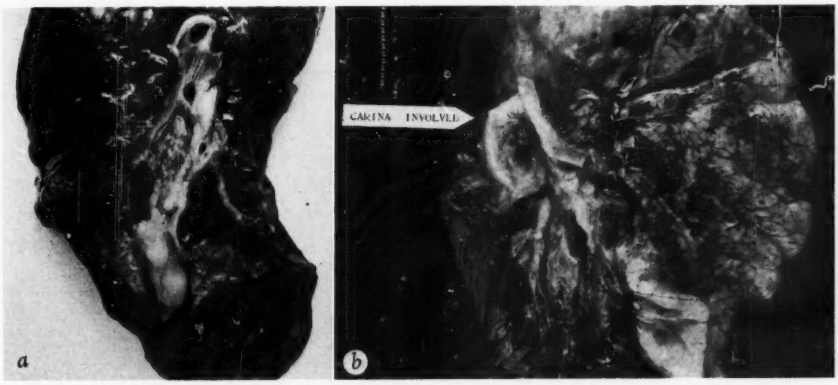

Fig. 4.—Specimens of lung from 2 patients with adenoid cystic carcinoma. *a*, Nodular and polypoid infiltrative tumor in right lower lobe bronchus (Case 7). *b*, Peribronchial thickening and carinal involvement by tumor (Case 12).

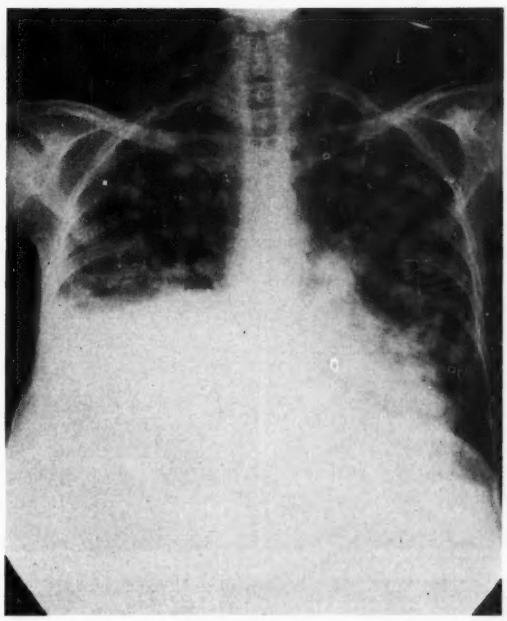

Fig. 5.—Extensive involvement of remaining lung 6 years after right middle and lower lobectomies for adenoid cystic carcinoma (cylindroma) (Case 11).

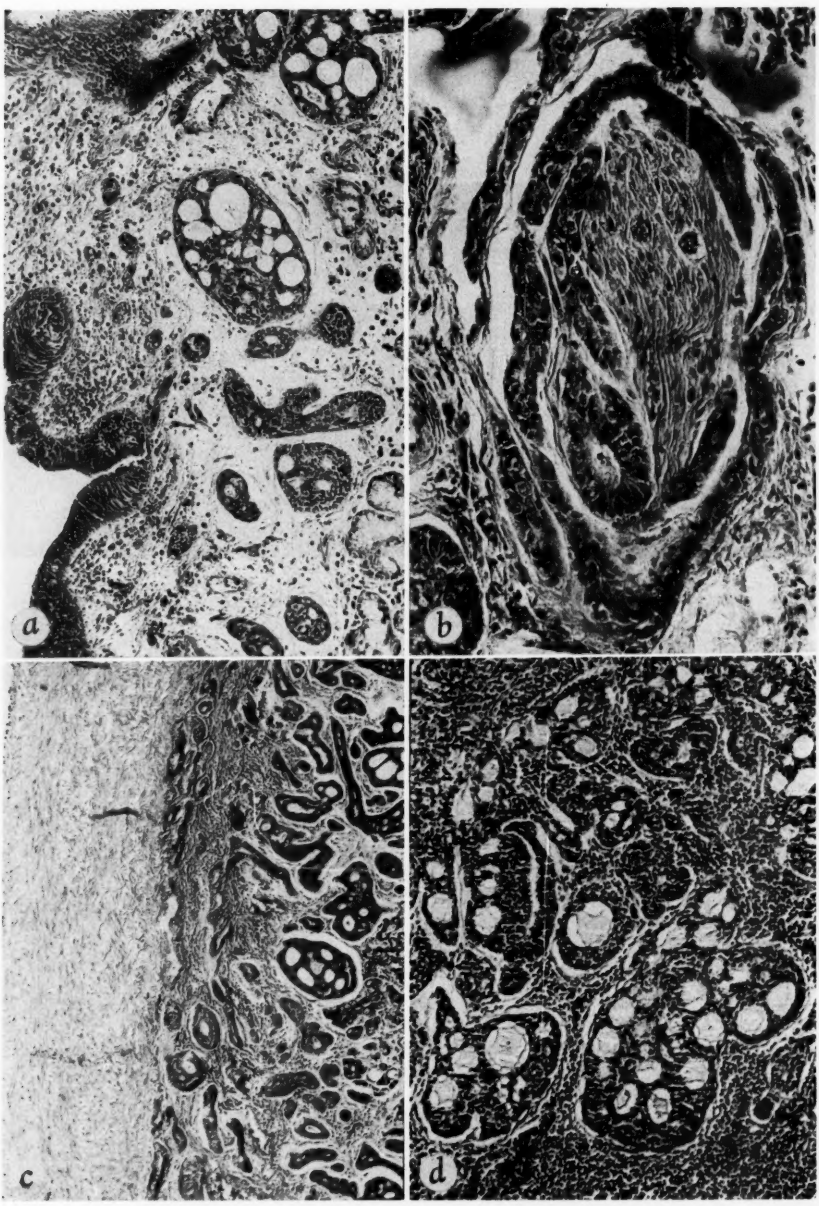

Fig. 6.—Adenoid cystic carcinoma. *a*, Mucosal extension at periphery of tumor. Note typical Swiss cheese pattern (hematoxylin and eosin; ×100). *b*, Perineural invasion (hematoxylin and eosin; ×300). *c*, Involvement of adventitia of large blood vessel (hematoxylin and eosin; ×50). *d*, Metastatic lesion in lymph nodes (hematoxylin and eosin; ×85).

reports or data obtained at the time of necropsy that four of the seven tumors showed gross involvement of mediastinal nodes, pericardium, or esophagus.

In addition to the six gross specimens of lung, material was available from necropsy on 2 patients treated by surgical resection. One (Case 7) who had died 10 months after pneumonectomy showed no evidence of residual tumor. The other (Case 11) had died 8 years after right middle and lower lobectomies, performed elsewhere. At the time of examination at the Mayo Clinic, extensive bilateral thoracic and hepatic involvement was apparent (Fig. 5) with jaundice and ascites. At necropsy, in addition to the thoracic involvement, extensive metastasis to the liver and peritoneum was found.

Microscopically, all of these lesions had the typical histologic features of adenoid cystic carcinoma. At the margins of the gross tumor there was extensive subepithelial infiltration of the mucosa (Fig. 6, *a*). In addition they all showed deep penetration and peribronchial extension with destruction of bronchial cartilages and invasion of neural elements (Fig. 6, *b*). Although tumor invasion

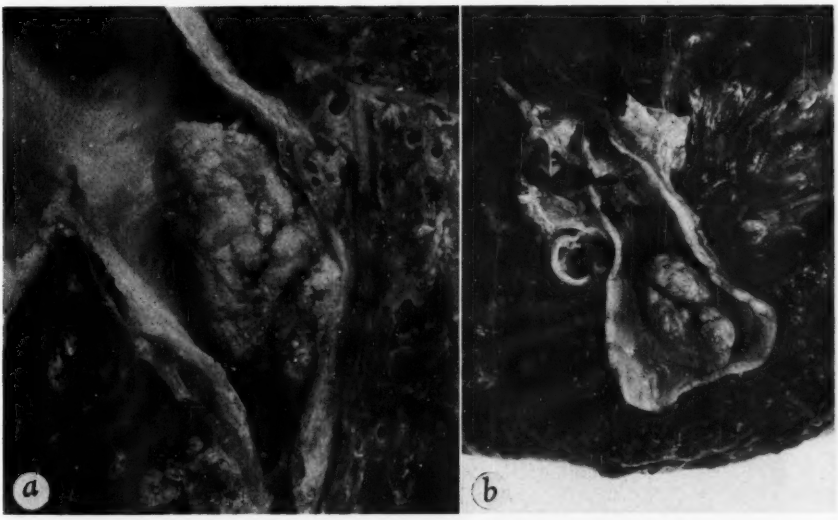

Fig. 7.—Two muco-epidermoid tumors. *a*, Postmortem specimen: right main bronchus viewed posteriorly (Case 14). *b*, Surgical specimen removed at pneumonectomy (Case 16).

of a vein was noted grossly in one specimen, none of the other six showed microscopic evidence of intravascular penetration, although all showed involvement of the adventitia and media of large blood vessels (Fig. 6, *c*). Peribronchial lymph nodes were involved by direct extension in four of the seven specimens and, in a fifth, embolic bronchial lymphatic metastasis had occurred (Fig. 6, *d*).

Five of the seven specimens of lung did not show gross involvement of the tracheobronchial junction, but on microscopic examination, tracheal extension was apparent in three. Thus, of the seven specimens of lung only two were free of tracheal extension.

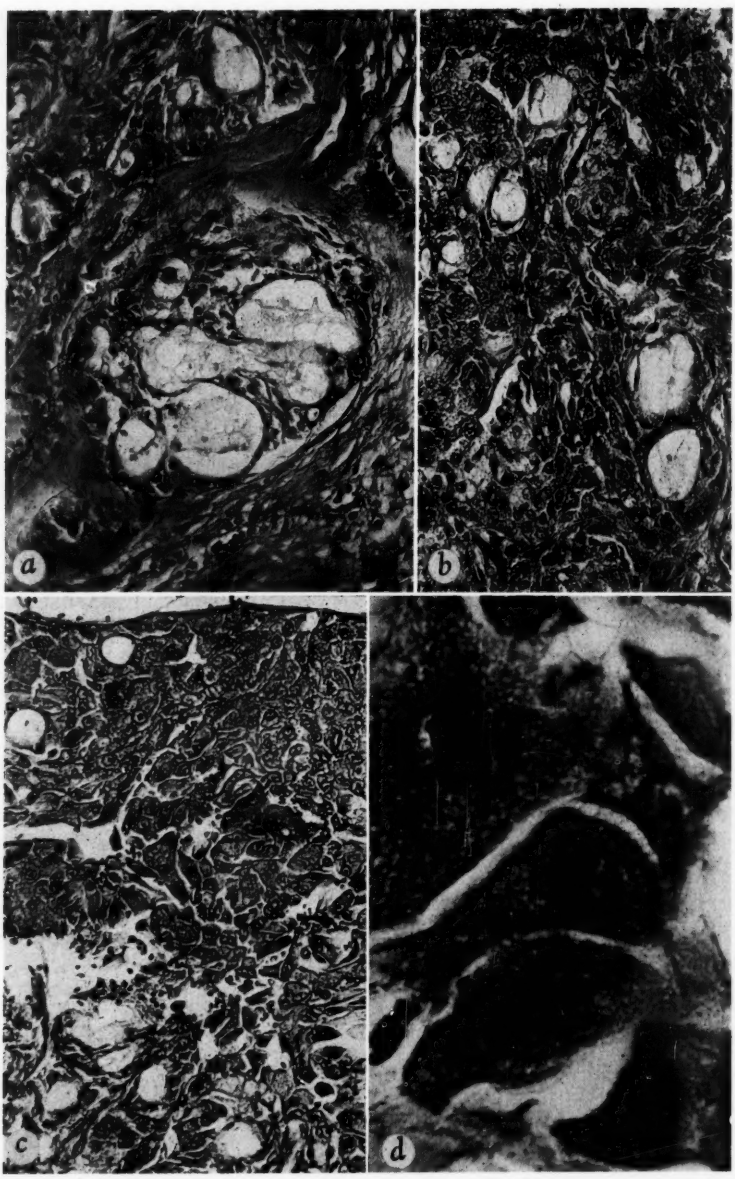

Fig. 8.—Muco-epidermoid tumor (hematoxylin and eosin). *a*, Two lines of differentiation should be noted (×300). *b*, Selected area with slightly less production of mucus (×300). *c*, Another tumor showing mainly squamous differentiation (×100). *d*, Same lesion as *c* showing intercellular bridges in squamous portion (×1,700). (This illustration has been reduced 1/10.)

Of further interest was the finding that the metastatic lesions of adenoid cystic carcinoma retain the typical histologic features of the primary tumor.

Muco-epidermoid Tumors.—In 2 of the 4 patients with muco-epidermoid tumors of the bronchus, surgical reports and surgically removed gross specimens of lung were available and data concerning the other 2 were obtained at the time of necropsy.

The distribution and gross form of these four tumors is illustrated in Fig. 3, *b*. The muco-epidermoid tumor in each of 3 cases appeared on gross inspection as a pedunculated polypoid endobronchial mass that completely occluded the bronchial lumen (Fig. 7). These tumors were covered with intact bronchial epithelium. As said before, the fourth muco-epidermoid tumor was situated extrabronchially and produced bronchial obstruction by extrinsic compression of the bronchus. None of the four muco-epidermoid tumors showed gross evidence of nodal extension or distant metastatic invasion.

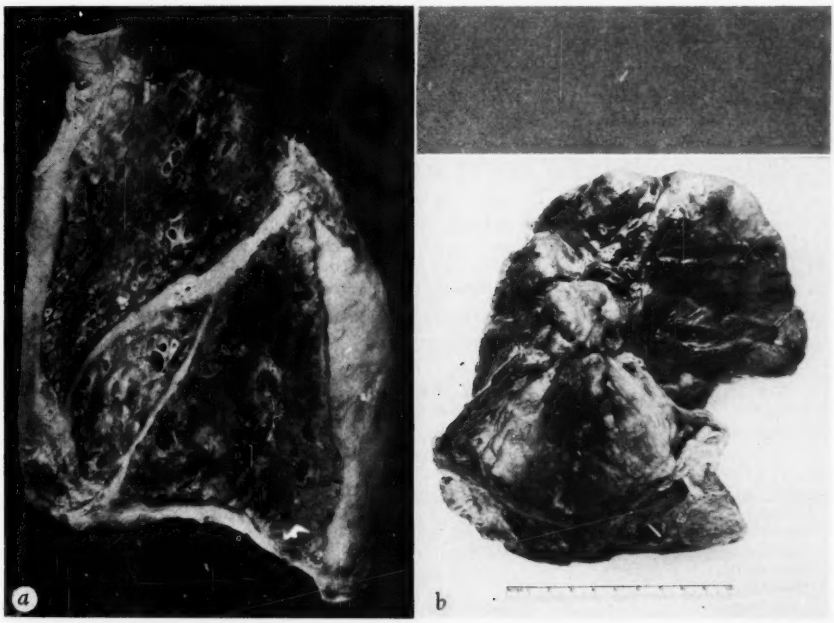

Fig. 9.—Suppurative pulmonary complications. *a*, Lung of patient with muco-epidermoid tumor showing extensive bronchiectasis and multiple pulmonary abscesses (Case 14). *b*, Resected lung of patient with adenoid cystic carcinoma showing large abscess in left lower lobe (Case 6).

Microscopic section showed these tumors to be covered with metaplastic squamous bronchial epithelium (Fig. 8). Sections through the site of attachment of endobronchial tumors to the bronchial wall showed extension of the tumor outward between, but not beyond, the bronchial cartilages with minimal infiltration at the edges in two of the three. The third endobronchial tumor extended to the bronchial cartilages, but not between or beyond. The extrabronchial muco-epidermoid tumor on microscopic examination was intimately associated with

deep bronchial glands. It did not encroach on cartilages nor did it have signs of infiltration about its margins. None of the four muco-epidermoid tumors showed destruction of cartilage or involvement of nerves, lymph nodes, blood vessels, trachea, mediastinal structures, or distant organs.

Pathologic Evidence for Pulmonary Suppuration.—The lungs of patients with adenoid cystic carcinoma and muco-epidermoid tumors showed the sequelae of bronchial obstruction. Bronchiectasis and obstructive pneumonitis were present in all specimens. In addition, four of the seven lungs of patients with adenoid cystic carcinoma and all four of those with muco-epidermoid tumors showed multiple abscess formation and extensive destruction of pulmonary tissue (Fig. 9). In one specimen pulmonary suppuration had progressed to the formation of bronchopleural fistula and chronic empyema (Case 15).

TREATMENT AND PROGNOSIS

In studying the prognosis in cases of adenoid cystic carcinoma and muco-epidermoid tumors of the bronchus, each group was divided into two subgroups, those in which treatment was surgical (pneumonectomy or lobectomy) and those in which treatment was more conservative (endoscopic surgical diathermy, radium, or roentgen ray).

Follow-up data in the form of one of the following was available during 1958 on all of the patients in this study: (1) recent examination at the Mayo Clinic, (2) reply to follow-up letters with accompanying recent thoracic roentgenogram, or (3) report of death. No patient was lost to follow-up.

Of the 13 patients who had primary adenoid cystic carcinoma, 7 underwent pulmonary resection. Six had pneumonectomy and 1 had right middle and lower lobectomies (Table V). The other 6 were treated conservatively because of the

TABLE V. RESULTS OF TREATMENT OF CYLINDROMA (ADENOID CYSTIC CARCINOMA) AND MUCO-EPIDERMOID TUMORS

TUMOR AND TREATMENT	TOTAL PATIENTS	RESULT		AVERAGE SURVIVAL (YEARS)
		ALIVE	DEAD	
Adenoid cystic carcinoma				
Resection	7	4	3	4.8
Conservative	6	0	6	2.8
Muco-epidermoid tumors				
Resection	2	2	0	9.0
Conservative	2	0	2	2.0

extent of their disease. The extent of the lesion of 1 patient did not permit resection at the time of surgical exploration; 4 had tracheal extension at the time of bronchoscopy and 1 had metastasis to the central nervous system at the time of diagnosis.

The survival of patients with adenoid cystic carcinoma following various forms of treatment is summarized in Table V. At the time of the follow-up study 4 of the 7 patients surgically treated were alive and well 1 to 13 years after treatment. The average duration of survival for this group was 5.2 years.

Three of the 7 patients treated by surgical resection were dead within 1 to 8 years. The average duration of survival for the 3 dead patients was 4.3 years following treatment.

None of the 6 patients treated conservatively for adenoid cystic carcinoma of the bronchus was alive at the time of follow-up. The duration of survival of these patients ranged from 1 week to 11 years following initial treatment.

The average duration of survival for all patients, both those living at time of follow-up and those dead, who had been treated by resection for adenoid cystic carcinoma of the bronchus, was 4.8 years. The average survival for all of those receiving conservative treatment was 2.8 years.

Two of the 4 patients with muco-epidermoid tumors of the bronchus were treated surgically, and 2 conservatively (Table V). Both patients treated surgically had pneumonectomies and both were living and well 8 and 10 years following treatment. Neither of the 2 patients treated conservatively was alive at the time of follow-up. One died 2 weeks after diagnosis and initial treatment; the other survived 4 years. Both of these patients had technically inoperable conditions, 1 because of acute sepsis and respiratory insufficiency and the other because of previous bronchopleural fistula, empyema, and thoracoplasty.

Certain considerations which influenced ultimate prognosis and reflected the nature of the tumors are worthy of comment. Sepsis and respiratory insufficiency secondary to bronchial obstruction and pulmonary suppuration were the major causes of death of patients treated by conservative means, irrespective of the type of tumor.

The other consideration influencing ultimate prognosis was the relative malignancy of the two types of tumor. In 3 of the 13 patients with adenoid cystic carcinoma, widespread metastasis developed. In only 2 of the 7 patients surgically treated was it possible to remove all the tumor. Only 1 of the 4 surviving patients with adenoid cystic carcinoma was believed to be free of tumor (Case 6). The remaining 3 had residual tumor, but all were in apparent excellent health at the time of follow-up, 1, 2½, and 4½ years after resection, and the thoracic roentgenograms showed no evidence of disease.

There was no evidence of distant spread in any of the patients with muco-epidermoid tumors. Although 2 showed rather minimal local invasion of the bronchus, none showed more distant extension. The 2 surviving patients were believed to have been cured of their tumor. The cause of death in the 2 patients treated conservatively was pulmonary suppuration. No evidence of metastasis was present at necropsy.

COMMENT

Adenoid cystic carcinoma behaves as a slowly growing, infiltrating malignant lesion; it invades adjacent structures in continuity, erodes cartilaginous tissue, and penetrates nerve structures and, occasionally, blood vessels. Having a prolonged and insidious course, the adenoid cystic carcinoma ultimately metastasizes distantly. Contrary to the findings in the more malignant bronchogenic carcinomas,[19] vascular invasion was not a significant feature.

Reid,[20] and Enterline and Schoenberg,[21] in an excellent review of adenoid cystic carcinoma of the bronchi and trachea, have confirmed the malignant nature of these tumors and have noted that they develop with equal frequency in both sites.

Clark, Clagett, and McDonald[22] reviewed 15 cases of primary tracheal cylindroma encountered at the Mayo Clinic. The cylindroma was found to be the second most common primary malignant lesion in the trachea and was characterized by slow growth and a prolonged course before the development of metastasis. Unfortunately, the results of both their study and the present one dealing with the bronchus show the inadequacies of present methods of dealing with primary tracheal tumors and the tracheal extension of bronchial tumors of this type. Of the 15 patients in their series who had tracheal cylindromas, 10 were dead at the time of the follow-up study. The majority of deaths were in patients treated by conservative means. Four patients were in good health at the time of follow-up, and all had had surgical excisions of their tracheal tumors.[22] Contrary to the conclusions of Vieta and Maier,[23] the results of these studies have not shown irradiation to be curative in any of these patients and have indicated that it is of questionable value in palliation.

Enterline and Schoenberg[21] made a selected collective review of 30 documented cases of cylindroma of the bronchus appearing in the literature. Nineteen patients were treated surgically. Sixty-three per cent were living free of disease, 21 per cent were living with tumor, and 15 per cent had died of their tumor at the time of the follow-up study. Of the 11 patients treated conservatively, 27 per cent were living and well, 27 per cent were living with tumor, and 45 per cent dead of tumor. Unfortunately, follow-up data in such studies are of limited value, but the figures do reflect the relative merit of pulmonary resection in palliation. It is inconceivable in the light of the present study to consider conservative therapy in any way curative.

Despite the poor ultimate prognosis of adenoid cystic carcinoma of the bronchus, pulmonary resection offers the best hope for cure and certainly the best chance for palliation. The bronchial obstructive sequelae seen in this series resembled that described by McBurney and associates[2] for carcinoid tumors. These complications alone were adequate reason for resection even if involved tissue was knowingly left behind. The evidence of suppurative disease was particularly impressive in the group treated conservatively. All but 2 of these patients succumbed to the suppurative complications of the disease before any evidence of metastasis became apparent. Although a number of patients gained intermittent relief of their bronchial obstruction over a period of years, none was improved beyond a state of pulmonary invalidism by conservative treatment. On the other hand, those patients whose lesions could be resected experienced immediate improvement. All of those surviving in spite of residual tumor are living active, useful lives.

We are aware of only 3 previous cases of muco-epidermoid tumors of the bronchus.[5, 24] The available data suggest that these neoplasms are of relatively low-grade malignancy, and none has spread beyond the bronchus. In the

present study these tumors produced disability by bronchial obstruction and secondary pulmonary suppuration. The two tumors amenable to surgical treatment were completely removed, and the problem of tracheal extension was not present. If the patient's condition permitted, surgical resection of the lung was performed. Local excision of the tumor was not indicated in any of our cases because of the presence of marked pulmonary suppuration distal to the tumor.

SUMMARY

In the 32 years, 1927 through 1958, 162 malignant tumors originating in the bronchial mucous glands were encountered at the Mayo Clinic. One hundred forty-five of these were carcinoid tumors, thirteen were cylindromas (adenoid cystic carcinoma), and four were muco-epidermoid tumors of the bronchus. The present study is concerned primarily with the adenoid cystic carcinomas and the muco-epidermoid tumors.

The presenting symptoms of all patients were predominantly related to bronchial obstruction and pneumonitis, and roentgenographic studies demonstrated some pulmonary collapse in most patients of both groups. Bronchoscopically, the adenoid cystic carcinoma appeared as an infiltrative malignant lesion, whereas the muco-epidermoid tumor predominantly appeared as a pedunculated polypoid mass. All involved major bronchi.

Pathologic data concerning the adenoid cystic carcinomas confirmed the bronchoscopic appearance as that of an infiltrative malignant lesion. These tumors were extensively invasive of peribronchial structures and often showed tracheal extension and mediastinal involvement. That these tumors are capable of widespread metastasis was demonstrated in a number of patients.

Similarly the pathologic data on the muco-epidermoid tumors paralleled their bronchoscopic appearance. Three were pedunculated polypoid endobronchial tumors and one produced bronchial obstruction by external compression. Two of the tumors showed some signs of local invasiveness, but none showed metastasis or extension beyond the point of bronchial attachment. Bronchial obstruction distal to the tumor frequently led to severe inflammatory sequelae regardless of the nature of the primary tumor. Often these suppurative complications were extreme.

Pneumonectomy was the only form of treatment that was presumably curative in patients with either type of tumor. In contrast to patients with muco-epidermoid tumors, only a few patients with adenoid cystic carcinoma had lesions that were potentially curable. Excellent palliation was possible when surgical resection was done in spite of the persistence of residual tumor. All patients in both groups who were treated by conservative means (endoscopic surgical diathermy, radium, or roentgen therapy) died. Most of them died of the suppurative complications of their disease.

REFERENCES

1. Moersch, H. J., and McDonald, J. R.: Bronchial Adenoma, J. A. M. A. **142**: 299-303, 1950.
2. McBurney, R. P., Kirklin, J. W., and Woolner, L. B.: Metastasizing Bronchial Adenomas, Surg. Gynec. & Obst. **96**: 482-492, 1953.

3. McBurney, R. P., Clagett, O. T., and McDonald, J. R.: Obstructive Pneumonitis Secondary to Bronchial Adenoma, J. THORACIC SURG. **24**: 411-419, 1952.
4. Goldman, A.: The Malignant Nature of Bronchial Adenoma, J. THORACIC SURG. **18**: 137-148, 1949.
5. Liebow, A. A.: Tumors of the Lower Respiratory Tract *in* Armed Forces Institute of Pathology, Atlas of Tumor Pathology, Section V, Fascicle 17, pp. 26-53, Washington, D. C., National Research Council, 1952.
6. Carlens, E., Wiklund, T., and Bergstrand, A.: Bronchial Adenoma: A Report of 70 Cases and a Critical Analysis of the Literature, Acta chir. scandinav. Suppl. 185, pp. 27-55, 1954.
7. Overholt, R. H., Bougas, J. A., and Morse, D. P.: Bronchial Adenoma: A Study of 60 Patients With Resections, Am. Rev. Tuberc. **76**: 865-884, 1957.
8. Knesvetich, E. M., McCormack, L. J., Effler, D. B., and Groves, L. K.: Bronchial Adenoma: A Clinicopathologic Study of 21 Cases, Cleveland Clin. Quart. **24**: 160-173, 1957.
9. Heschl, H.: Ueber ein Cylindrom der Lunge, Wien. med. Wchnschr. **27**: 385-390, 1877.
10. Müller, H.: Quoted by Engelbreth-Holm, J.: Benign Bronchial Adenomas, Acta chir. scandinav. **90**: 383-409, 1945.
11. Kramer, R.: Adenoma of the Bronchus, Ann. Otol., Rhin. & Laryng. **39**: 689-695, 1930.
12. Geipel, P.: Zur Kenntnis der gutartigen Bronchialtumoren, Frankfurt. Ztschr. Path. **42**: 516-544, 1931.
13. Wessler, H., and Rabin, C. B.: Benign Tumors of the Bronchus, Am. J. M. Sc. **183**: 164-180, 1932.
14. Hamperl, H.: Über gutartige Bronchialtumoren (Cylindrome und Carcinoide), Arch. path. Anat. **300**: 46-88, 1937.
15. Billroth, T.: Beobachtungen über Geschwülste der Speicheldrüsen, Arch. path. Anat. **17**: 357-375, 1859.
16. McDonald, J. R., Moersch, H. J., and Tinney, W. S.: Cylindroma of the Bronchus: Report of Six Cases, J. THORACIC SURG. **14**: 445-453, 1945.
17. Foote, F. W., Jr., and Frazell, E. L.: Tumors of the Major Salivary Glands, *in* Armed Forces Institute of Pathology, Atlas of Tumor Pathology, Section IV, Fascicle 11, pp. 103-115, Washington, D. C., National Research Council, 1954.
18. Stewart, F. W., Foote, F. W., and Becker, W. F.: Muco-epidermoid Tumors of Salivary Glands, Ann. Surg. **122**: 820-844, 1945.
19. Ballantyne, A. J., Clagett, O. T., and McDonald, J. R.: Vascular Invasion in Bronchogenic Carcinoma, Thorax **12**: 294-299, 1957.
20. Reid, J. D.: Adenoid Cystic Carcinoma (Cylindroma) of the Bronchial Tree, Cancer **5**: 685-694, 1952.
21. Enterline, H. T., and Schoenberg, H. W.: Carcinoma (Cylindromatous Type) of Trachea and Bronchi and Bronchial Adenoma: A Comparative Study, Cancer **7**: 663-670, 1954.
22. Clark, P. L., III, Clagett, O. T., and McDonald, J. R.: Cylindromas of the Trachea, Proc. Staff Meet. Mayo Clin. **28**: 513-519, 1953.
23. Vieta, J. O., and Maier, H. C.: The Treatment of Adenoid Cystic Carcinoma (Cylindroma) of the Respiratory Tract by Surgery and Radiation Therapy, Dis. Chest **31**: 493-511, 1957.
24. Liebow, A. A.: Personal communication to the authors.

(For Discussion, see page 739.)

BRONCHOGENIC CARCINOMA

An Aggressive Surgical Attitude

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INTRODUCTION

THE SURGEON who recommends a thoracotomy for bronchogenic carcinoma implies that the disease can be cured by resection. However, the recent medical literature reports that in about 40 per cent of operated cases, resection could not or should not be performed. This discrepancy between intent and accomplishment—tragic to the patient—presents a challenge to surgical skill and judgment. Consequently, in the last 10 years we have gradually extended our technical efforts to perform resection in more and more far-advanced cases.

The justification for this surgical attitude was based not only upon modern technical advances but, also, upon the favorable biologic behavior of the lesion which permitted extensive growth locally, yet without detectable systemic dissemination. Somewhat to our surprise, the morbidity and mortality rates remained acceptably low and, as a result of our determined effort to increase the resectability rate, a few additional patients appear to be cured and many lives prolonged.

DEFINITIONS, CLASSIFICATION, AND DESCRIPTION OF OPERATIONS

The surgical aspects of the literature on bronchogenic carcinoma are often difficult to evaluate because of ambiguous terminology. Various authors have used such terms as "operable," "resectable," "radical resection," "palliative," "curative," etc., in different contexts.

The term "resection," by definition, means the removal of a considerable portion of an organ, in contrast to removal of an entire organ. However, this term is in popular usage in both contexts, and in this report it is used to denote removal of a portion of a lung, an entire lung, and any other structures included in the surgical specimen.

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We recommend the use of the following terms in defining operative categories.

1. Standard resection.
2. Extended resection.
3. Noncurative resection.
4. Palliative resection.
5. Explored only.

These operations are defined in more detail below.

1. *The "Standard Resection."*—Patients in this category were considered at operation to be *potentially cured* and the surgical specimen consisted only of pulmonary structures, parietal pleura when adherent, and mediastinal lymphatic tissue. Bronchoplastic procedures and resections of the tracheal carina were the only exceptions to these criteria and were not included in this category.

Mediastinal lymph node dissection was usually performed in the standard resection but not of the en bloc type. We arbitrarily divided this maneuver into upper and lower mediastinal dissections. The lower dissection was most often performed, and consisted of the removal of the lymph nodes inferior to the azygos vein on the right side and to the aortic arch on the left. It included periaortic, periesophageal, subcarinal, distal retrotracheal, and peribronchial nodes from the opposite main-stem bronchus. The upper dissection was of the mediastinal lymph nodes along the trachea and superior vena cava; these were removed whenever there was suspicion that they were involved and whenever the primary tumor was adjacent.

2. *The "Extended Resection."*—Patients in this category were also considered at operation to be *potentially cured.* Complete resection in these cases required removal of extrapleural structures invaded by carcinoma. The surgical specimens therefore included various combinations of pulmonary structures, portions of trachea, pericardium, atrium, intrapericardial vessels, chest wall, diaphragm, and esophagus. A mediastinal lymph node dissection, as described above under "standard resection," was done routinely when the "extended" procedure was carried into the mediastinum. Bronchoplastic procedures and partial resections of the trachea were included in this category.

3. *The "Noncurative Resection."*—Patients in this category were considered to be *potentially curable preoperatively,* had resection, but at operation were found to have additional inaccessible carcinoma. Frequently, this observation could be made only after removal of the major specimen. However, sometimes the observation of surgical incurability could be made immediately upon opening the hemithorax, yet removal of the major lesion was considered justifiable to avert secondary complications such as obstructive pneumonitis, abscess, obstructive emphysema, pain, and hemorrhage. Furthermore, it was considered advantageous to remove the major lesion so that radiation therapy could be confined to the mediastinal contents and thus avoid radiation complications in the residual lung.

The "noncurative resection" group was subdivided into the "standard" and "extended" procedures for the purpose of compiling operative complications and mortality rates. However, it seemed illogical to subdivide this group for the assessment of survival statistics, since none of the patients was considered to be potentially cured at operation.

4. *The "Palliative Resection."*—Patients in this category were known to be *incurable prior to thoracotomy* because of remote metastases, pleural effusion containing malignant cells, contralateral pulmonary metastases, or other unequivocal criteria of incurability. Resections were performed in this group *not* to treat bronchogenic carcinoma per se, but to palliate symptoms such as intractable cough, pain, hemorrhage, and uncontrollable infection that were secondary to the presence of the carcinoma.

We reasoned that the "noncurative" resections should not be included in the "palliative" category, since many of these patients did not have symptoms requiring immediate palliation, and, furthermore, they were considered to be potentially curable preoperatively in contrast to the palliative group as defined.

5. *The "Explored Only" Category.*—Patients included in this category were also considered to be *potentially curable preoperatively.* However, resection was not done in this group either because the findings were such that an early fatal outcome could be predicted before secondary local complications developed, or because the widespread dissemination of carcinoma precluded effective radiation therapy (diffuse pleural implants).

This report concerns 248 thoracotomies that are grouped according to the above operative categories. The operations performed and their relative frequencies are recorded in Table I.

TABLE I. OPERATIVE CATEGORIES (248 CASES)

TYPE OPERATION	NUMBER	PER CENT	PER CENT RESECTED	PER CENT POTENTIALLY CURATIVE
Standard resection	120	48.4	77	61.7
Extended resection	33	13.3		
Noncurative resection	32	12.9		
Palliative resection	6	2.4		
Explored only	57	23.0		
Totals	248	100.0		

CASE MATERIAL

The 248 cases of bronchogenic carcinoma included in this report were private patients of the senior author (J. M. C.) and in each instance he was the operating surgeon. All patients who had a thoracotomy for this disease during the years 1949-1958 are included, except for 3 or 4 cases in which there was no tissue diagnosis. During this 9-year period approximately twice this number of thoracotomies for bronchogenic carcinoma was performed, either by the senior author in a consulting capacity or by resident or attending surgeons whom he assisted. These cases were excluded to avoid unpredictable variables related to technique.

The positive tissue diagnosis in each case was obtained by bronchoscopic biopsy, thoracotomy biopsy, or by resection of the lesion. The histologic diagnosis was based upon the most predominant cell type. The types and their

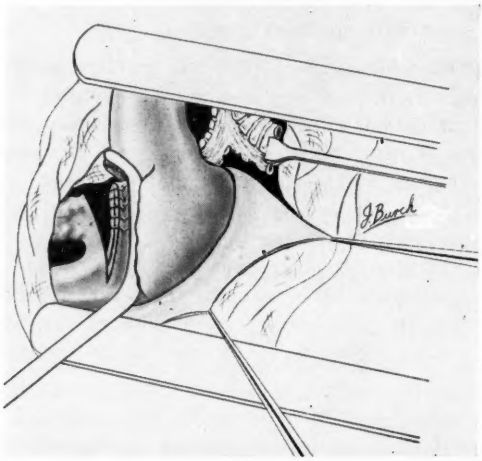


Fig. 1.—Resection of the left auricle in continuity with the right pulmonary veins. Carcinoma is shown invading the superior vein.

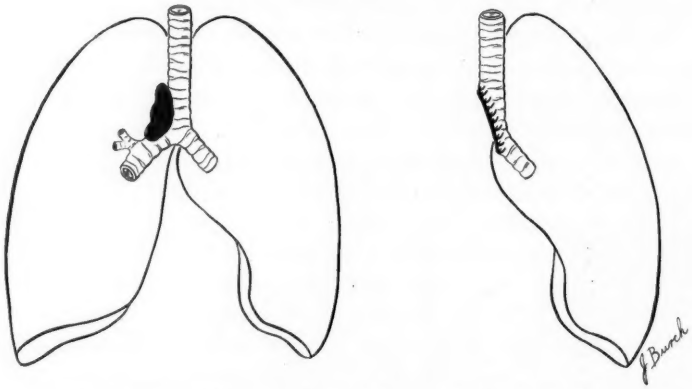


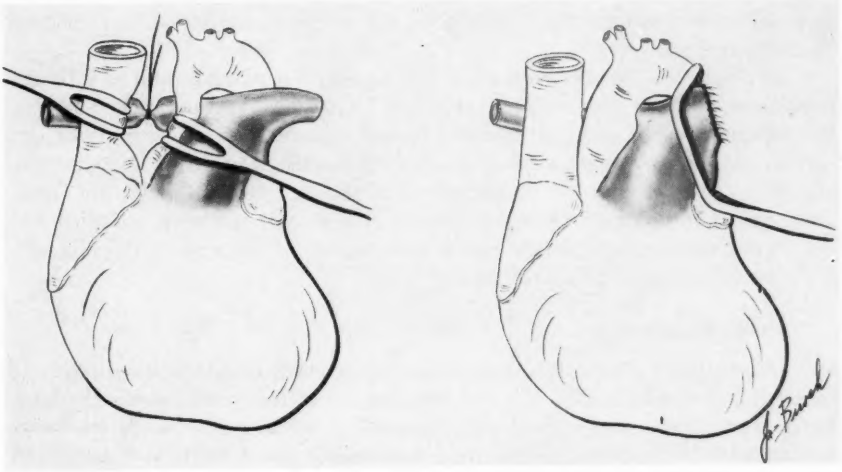
Fig. 2.—Resection of the tracheal carina and the lateral tracheal wall, with primary closure.

relative frequencies were epidermoid carcinoma (54 per cent), adenocarcinoma (16 per cent), anaplastic carcinoma (24 per cent), and bronchiolar carcinoma (6 per cent). The latter group was included in this report since the bronchiolar origin is generally accepted by pathologists and since the criteria for operation were the same as those in the other histologic categories.

The sex incidence of bronchogenic carcinoma in this series was 222 in the male and 26 in the female, a ratio of 8.5:1. There was a relative preponderance of adenocarcinoma and bronchiolar carcinoma in the female. The mean age for males in this series was 57 and for females 51 years.

THE EXTENDED OPERATION

To encompass both the visible and palpable limits of the lesion it was necessary in some cases to perform an extended operative procedure by resecting the pericardium, atrial wall (Fig. 1) and, occasionally, if necessary,



A. *B.*

Fig. 3.—*A*, The superior vena cava and the ascending aorta are retracted to permit proximal ligation of the right main pulmonary artery. *B*, Partial occlusion of the pulmonary trunk to permit amputation of the left main pulmonary artery at its origin.

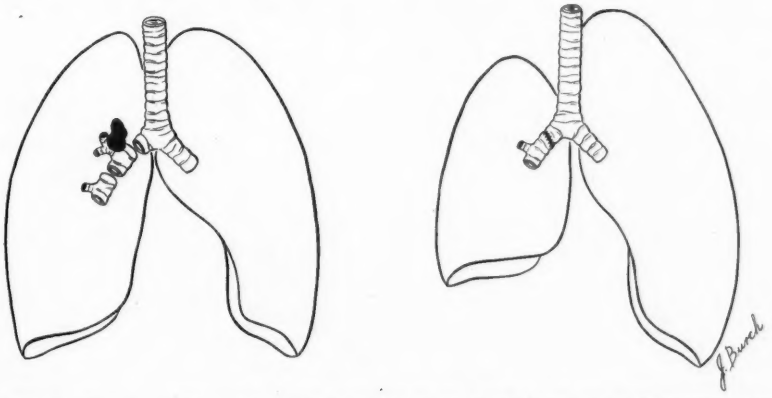


Fig. 4.—Sleeve resection of the main bronchus in continuity with upper lobectomy. Anastomosis of the main and intermediate bronchi is shown, preserving essential pulmonary function.

the tracheal carina (Fig. 2). If the pulmonary artery was involved it was necessary to divide it at its origin (Fig. 3). In a few instances when the respiratory reserve was critically low, a sleeve resection of the main bronchus was included with the upper lobe. The intermediate bronchus then was

anastomosed to the stump of the main bronchus in order to preserve pulmonary function (Fig. 4). On occasion we have found it necessary to include with the specimen a portion of the chest wall or the superior vena cava, and others who have reported on this subject have resected a portion of the diaphragm, a branch of the brachial plexus, short segments of the superior vena cava, the pulmonary artery, and even the aorta. We believe that all such maneuvers should be included in the armamentarium of the modern thoracic surgeon.

As a rule, we did not recommend thoracotomy for patients who had distant metastases, superior vena caval obstruction, involvement of the brachial plexus, left recurrent nerve paralysis, obvious cardiac failure, respiratory insufficiency, and uncontrollable extrathoracic systemic disease, such as nephritis and cirrhosis. However, the presence of bloody pleural effusion without malignant cells, paralysis of the phrenic nerve, or history of a proved coronary occlusion was not, in our opinion, an absolute contraindication to an exploratory thoracotomy. Such cases required individualization.

OPERATIVE MORBIDITY

The complete morbidity of a surgical procedure is sometimes difficult to assess, for it depends not only on the effect of trauma inflicted on the body but also on a subjective response, in the mind of the patient. The latter factor is intangible, but is common to every operation. We believe, therefore, that the mean duration of hospital stay and the incidence of major complications give as reliable an indication as can reasonably be obtained of any trend in morbidity that the "extended" operation might produce. Table II shows that the mean duration of postoperative hospital stay was not increased significantly by extending the operation.

TABLE II. BRONCHOGENIC CARCINOMA
POSTOPERATIVE DAYS IN HOSPITAL

Explored only	12.5
"Standard" resection	14.4
"Extended" resection	15.4
Palliative resection	14.2

Major operative complications encountered in this series of 248 cases, including the 23 which were fatal, are arranged in Table III according to the type of operation. There were five bronchopleural fistulas, three following right pneumonectomy and two following left upper lobectomy. Two of these responded to conservative management and two required thoracoplasty. The fifth was fatal. Nonfatal empyema without demonstrable bronchial fistula occurred twice, and in only 1 case was thoracoplasty required. Two additional cases were fatal. One wound infection occurred in the entire series (0.4 per cent). Postoperative hemorrhage that required re-exploration for its control occurred three times. Fatal hemorrhage at operation occurred once. Only 2 patients had a postoperative myocardial infarction, 1 is still living 2½ years after an "extended" pneumonectomy that included pericardium involved

by carcinoma, and the other patient died. The additional complications that occurred in this series were fatal. These are described in the section on operative mortality.

TABLE III. OPERATIVE COMPLICATIONS

COMPLICATION	TYPE OF OPERATION*			TOTAL (248)
	STANDARD (142)	EXTENDED (43)	EXPLORED (57)	
Cardiac arrest	1	0	0	1
Herniation of the heart	0	1	0	1
Hemorrhage	2	2	0	4
Bronchopleural fistula	5	0	0	5
Empyema	2	2	0	4
Wound infection	0	1	0	1
Bronchopneumonia	1	1	0	2
Cardiorespiratory failure	5	1	2	8
Pulmonary embolus	1	2	0	3
Myocardial infarction	1	1	0	2
Metastases	1	2	0	3
Totals	19	13	2	34
Per cent	13.4	30.2	3.5	13.7

*No complication in palliative group (6 cases).

The relative frequency of complications in the "standard" and "extended" operations is also demonstrated in Table III. The numbers of each complication were too small to exclude fortuitous occurrences, however, no condemning incidence was noted in the "extended" group. The complication rate for the "extended" operations was 30.2 per cent compared with 13.4 per cent for the "standard" operations. No complication occurred among the 6 cases of "palliative resection." Two fatal complications occurred among the 57 explored cases (3.5 per cent). The over-all complication rate for the 248 cases in this report therefore was 13.7 per cent.

OPERATIVE MORTALITY

Operative death in this report is defined as death occurring from any cause during operation or while still in the hospital postoperatively.

Hospital deaths numbered 23 among the 248 operations, a total mortality rate of 9.3 per cent. Table IV lists the causes of death and the respective mortality rates according to the type of operation. Three deaths occurred during operation, one due to herniation of the heart after partial pericardiectomy, one due to exsanguination that occurred when lung retraction avulsed tumor which comprised the right atrial wall, and the third was due to cardiac arrest.

Bronchopleural fistula complicated by pneumonia caused death in 1 case following a "standard" left upper lobectomy. Two patients died after a "standard" resection with empyema, complicated by septicemia in 1 case and in the other by enterocolitis.

Eight deaths were attributed to cardiac or respiratory insufficiency. Only 1 of these patients had an "extended" operation. Bronchopneumonia

following pneumonectomy was fatal to 2 patients, 1 in each of the resected categories. Three patients died from pulmonary embolus, 2 of whom had "extended" surgery. Fatal myocardial infarction, 5 days after a "standard" pneumonectomy, occurred once.

TABLE IV. OPERATIVE MORTALITY

CAUSES OF DEATH	TYPE OF OPERATION* STANDARD (142)	EXTENDED (43)	EXPLORED (57)	TOTAL (248)
Herniation of the heart	0	1	0	1
Cardiac arrest	1	0	0	1
Exsanguination	0	1	0	1
Bronchopleural fistula	1	0	0	1
Empyema	2	0	0	2
Cardiorespiratory failure	5	1	2	8
Bronchopneumonia	1	1	0	2
Pulmonary embolus	1	2	0	3
Metastases	1	2	0	3
Myocardial infarction	1	0	0	1
Totals	13	8	2	23
Per cent	9.1	18.6	3.5	9.3

*No mortality in palliative group (6 cases).

Distant metastases became evident postoperatively in 3 cases. In 1 of these, the bowel ruptured at the site of serosal implantation and death was caused by peritonitis. Carcinomatosis was fatal 1 month postoperatively for 1 patient and metastases became evident in the brain of another and caused death after 6 weeks.

The relative occurrence of hospital deaths in the "standard," the "extended," and the "explored" categories is listed in Table IV. Thirteen deaths among the 142 "standard" resections represented a mortality rate of 9.1 per cent, whereas the 8 deaths among the 43 patients having "extended" resections represented a mortality rate of 18.6 per cent. These deaths, plus the 2 among the 57 explored cases (3.5 per cent), comprise a total mortality of 9.3 per cent for the 248 operations. No hospital deaths occurred among the 6 incurable patients whose operations were performed only for palliation of symptoms.

The first five causes of death listed in Table IV were considered to be directly attributable to the type of operation performed. Table V lists operative deaths due to surgical technique and other causes for each type of operation. This will be referred to in the final discussion.

TABLE V. OPERATIVE MORTALITY

OPERATION	SURGICAL TECHNIQUE (PER CENT)	OTHER CAUSES (PER CENT)	TOTAL (PER CENT)
Standard resection (142)	2.1	7.0	9.1
Extended resection (43)	4.6	14.0	18.6
Explored only (57)	0.0	3.5	3.5
Totals* (248)	2.0	7.3	9.3

*No mortality in palliative group (6 cases).

SURVIVAL

Just as the total operative morbidity of any surgical procedure is incompletely assessed from a statistical aspect alone, so are survival statistics following thoracotomy for bronchogenic carcinoma only a partial indication of its over-all merit. Those patients whose carcinomas were amenable to potentially curative resection and who survived without radiation therapy for at least 3 years with no recurrence, probably survived as a result of the operation. These cases are the ultimate reward of a surgeon's efforts, and should make every thoracic surgeon operating on bronchogenic carcinoma determined to accomplish as many potentially curative resections as possible. In this report, 13.3 per cent of all our patients achieved a "potentially curative" status by means of an "extended resection" (Table I). The postoperative follow-up period of this series is too brief at present to permit even a 3-year appraisal of the majority of "extended resections." However, general trends of survival could be determined and are presented in Table VI.

TABLE VI. SURVIVAL

OPERATION	NUMBER LEAVING HOSPITAL*	NUMBER LIVING	NUMBER 3-8 YEAR SURVIVORS	MEAN SURVIVAL (MO.)
Standard resection	110	40	22	21.6
Extended resection	26	13	2	16.7
Noncurative resection	28	1	1†	11.5
Palliative resection	6	0	0	4.4
Explored only	55	3	2†	8.8

*Excludes operative deaths.
†Radiation therapy.

The patients who had a "standard resection" have a mean survival of 21.6 months to date; 36.7 per cent of these patients are still living and remain potentially cured. The patients who had an "extended resection" have a mean survival of 16.7 months to date; 50 per cent of these patients are still living and are potentially cured. There are 22 3-year survivors of the "standard resection" group and only 2, thus far, in the "extended resection" group. A major factor in the differences in these figures is the more recent introduction of the "extended resection."

The comparable survival statistics for patients not potentially cured at operation are impressively low in each category. Of 28 patients in the "noncurative" resection group, only 1 patient is living, and he is known to have diffuse contralateral lung metastases. Only 1 patient in this group survived longer than 3 years, and he succumbed to carcinomatosis after 4 years and 2 months. The mean survival following "noncurative resection" was 11.5 months. Most of these patients received radiation therapy postoperatively.

The patients who had exploratory operations without resection survived an average of 8.9 months. The 3 survivors in the "explored only" group are especially interesting since they have no counterpart in the "noncurative resection" group. These 3 patients are males who had right upper lobe infiltrating carcinomas. They received radiation therapy and remain clinically well

without evidence of metastases or increasing local growth. They have survived 4 years, 3 years and 7 months, and 1 year, respectively.

DISCUSSION

The increasing incidence of bronchogenic carcinoma in this country fortunately has occurred during an era of rapid advances in thoracic surgical technique. The recent trend toward earlier diagnosis has increased the chances of successful surgery in many cases. However, the reported number of patients who were *not* potentially cured by surgery remains high. This report concerns especially the use of an *"extended resection"* for patients who would have been incurable by standard lung resection techniques.

Carcinoma in any organ may behave in unpredictable ways. When one considers that lung tissue is made up entirely of blood, lymphatic and air channels, it would seem especially remarkable that a lung carcinoma could ever grow large *without* clinical evidence of systemic dissemination. Yet this is often the case, and this phenomenon must indicate the relative benignity of these extensive local growths. Perhaps such cases deserve especially the application of every available thoracic surgical technique to effect a potentially curative resection.

We believe that there should be a *universally accepted nomenclature* that would portray the accomplishments of the surgeon, and which should not be determined or influenced by the delayed but final report of the pathologist. This qualification would seem practical since the surgeon decides the dimensions of the operation at the operating table without microscopic control to define the limits of the carcinoma. The terminology for operative categories submitted in this report permits clear-cut allocation of each case, and permits relative assessment of the maximum surgical efforts to cure bronchogenic carcinoma.

We did not consider it useful to include in the terminology reference to the amount of lung tissue removed, i.e., pneumonectomy, lobectomy, and segmental resection. These operative terms more often reflect the fortuitous position and nature of the primary lesion, rather than the surgeon's accomplishment in removing all carcinoma possible.

The terms "standard resection" and "extended resection," according to the definitions presented, include the only patients who were considered to be potentially curable at the time of operation. These cases alone, therefore, represent surgical accomplishment, and this might be best expressed in terms of a *"potentially curative resection rate."* This was 61.7 per cent for this series of 248 cases. The total resectability rates (77.0 per cent in this report) that usually are recorded in the literature are meaningless from the standpoint of surgical accomplishment, since usually there is no indication of how many noncurative resections were included.

"Extended resection" techniques achieved, in this series, a potentially curative status for 26 patients. These patients would have been incurable by standard techniques of pulmonary resection. The operative complications and mortalities incurred to establish this group were approximately twiee the rates for

the "standard resection." The mortality rates consequent to the "standard" and the "extended resections" were 9.1 and 18.6 per cent, respectively (Table IV). However, even these rates were acceptably low, especially when the complications that were fatal and directly attributable to surgical technique were considered separate from those causes of death that might be expected following any major operation (Table V). Listed in this fashion, mortality rates were 2.1 per cent and 4.6 per cent, respectively, for the "standard" and the "extended resections."

Continued experience with bronchial and cardiovascular techniques should permit fewer complications among the "extended resections." We have learned, for example, to close large pericardial defects with parietal pleura to prevent herniation of the heart, and to use electrocoagulation almost routinely just prior to closing the thorax for more complete hemostasis in the extrapleural planes and on raw lung surfaces. Careful observance of the basic principles of bronchial closure was essential to maintain a low incidence of bronchopleural fistula.

A note of caution is indicated for the bronchoplastic procedures in which sleeve resection and bronchial anastomosis were performed to preserve lower lobe function. We consider that this procedure should be used only when the respiratory reserve is such that pneumonectomy could not possibly be tolerated.

Several years must pass before the customary 5-year survival data can be compiled for this series of cases. The trends that are apparent in Table VI constitute sufficient evidence, in our opinion, to state that the "extended resection" not only is justifiable but definitely is indicated whenever it is technically possible. The alternative for these patients is to abandon hope entirely, and to deny them and their families the satisfaction that everything possible surgically was done.

SUMMARY

In the surgical treatment of bronchogenic carcinoma, it is not our intent to encourage unguarded aggressiveness in the modern thoracic surgeon. During the past 25 years the indications for and against thoracotomy have been fairly well standardized. The 248 cases in this report probably would have been selected for operation by the majority of experienced thoracic surgeons.

The biologic behavior of bronchogenic carcinoma is often paradoxical; it may become extensive and invasive locally without detectable systemic dissemination. In our opinion, such lesions deserve an extended surgical effort to accomplish complete removal.

Twenty-six patients in this series were afforded a potentially curative status by extended surgical efforts without unduly increasing the operative morbidity and mortality.

The favorable survival pattern in this group encourages us to recommend this aggressive surgical attitude.

Once a patient has accepted our surgical recommendation, we are obliged to justify his confidence by including in our surgical armamentarium the techniques outlined.

REFERENCES

Abbott, O. A.: Experiences With the Surgical Resection of the Human Carina, Tracheal Wall, and Contralateral Bronchial Wall in Cases of Right Total Pneumonectomy, J. THORACIC SURG. **19**: 906, 1950.

Abbott, O. A., Van Fleit, W. E., and Roberto, A. E.: Experiences With Extending the Indications for the Use of Tracheal and Bronchial Grafts, J. THORACIC SURG. **29**: 217, 1955.

Allison, P. R.: Discussion of Surgical Management of Carcinoma of the Lung, J. THORACIC SURG. **20**: 349, 1950.

Auerbach, O., Gere, J. B., Pawlowski, J. M., Muehsam, G. E., Smolin, H. J., and Stout, A. P.: Carcinoma-In-Situ and Early Invasive Carcinoma Occurring in the Tracheobronchial Trees in Cases of Bronchial Carcinoma, J. THORACIC SURG. **34**: 298, 1957.

Aylwin, J. A.: Avoidable Vascular Spread in Resection for Bronchial Carcinoma, Thorax **6**: 250, 1951.

Ballantyne, A. J., Clagett, O. T., and McDonald, J. R.: Vascular Invasion in Bronchogenic Carcinoma, Thorax **12**: 294, 1957.

Barclay, R. S., McSwan, N., and Welsh, T. M.: Tracheal Reconstruction Without the Use of Grafts, Thorax **12**: 177, 1957.

Belsey, R.: Stainless Steel Wire Suture Technique in Thoracic Surgery, Thorax **1**: 39, 1946.

Belsey, R.: Resection and Reconstruction of Intrathoracic Trachea, Brit. J. Surg. **38**: 200, 1950.

Bettman, R. B., and Tannenbaum, W. J.: Herniation of the Heart Through a Pericardial Incision, Ann. Surg. **128**: 1012, 1948.

Blades, B.: Presidential Address—A Case Report and Miscellaneous Comments, J. THORACIC SURG. **36**: 285, 1958.

Boyd, D. P., Smedal, M. I., Kirtland, H. B., Jr., Kelley, G. E., and Trump, J. G.: Carcinoma of the Lung; A Report of 403 Cases, J. THORACIC SURG. **28**: 392, 1954.

Brewer, L. A., III., Bai, A. F., Little, J. N., Gregorio Rabago y Pardo: Carcinoma of the Lung, J. A. M. A. **166**: 149, 1958.

Brock, R. C.: Bronchial Carcinoma, Brit. M. J. **2**: 737, 1948.

Burford, T. H., Center, S., Ferguson, T. B., and Spjut, H. J.: Results in the Treatment of Bronchogenic Carcinoma, J. THORACIC SURG. **36**: 316, 1958.

Burnett, W. E., Rosemond, G. P., Hall, J. H., and Caswell, H. T.: Carcinoma of the Lung, Arch. *Surg.* **61**: 1052, 1950.

Cahan, W. G., Watson, W. L., and Pool, J. L.: Radical Pneumonectomy, J. THORACIC SURG. **22**: 449, 1951.

Churchill, E. D., Sweet, R. H., Scannell, J. G., and Wilkins, E. W., Jr.: Further Studies in the Surgical Management of Carcinoma of the Lung, J. THORACIC SURG. **36**: 301, 1958.

Churchill, E. D., Sweet, R. H., Soutter, L., and Scannell, J. G.: Surgical Management of Carcinoma of the Lung, J. THORACIC SURG. **20**: 349, 1950.

Cotton, B. H., and Penido, J. R. F.: Pleuropulmonary Resection With Hemidiaphragmectomy. Preliminary Report, J. THORACIC SURG. **22**: 474, 1951.

Crafoord, C.: Personal communication.

Davis, E. W., Peabody, J. W., Jr., and Katz, S.: The Solitary Pulmonary Nodule, J. THORACIC SURG. **32**: 728, 1956.

Ehler, A., Stranahan, A., and Olson, K. B.: Bronchogenic Carcinoma, New England J. Med. **251**: 207, 1954.

Gebauer, P. W.: Plastic Reconstruction of Tuberculous Bronchostenosis With Dermal Grafts, J. THORACIC SURG. **19**: 604, 1950.

Gebauer, P. W.: Further Experiences With Dermal Grafts for Healed Tuberculous Stenosis of the Bronchi and Trachea, J. THORACIC SURG. **20**: 628, 1950.

Gibbon, J. H., Templeton, J. Y., and Nealson, T. F.: Factors Which Influence the Long Term Survival of Patients With Cancer of the Lung, Ann. Surg. **145**: 637, 1957.

Grindlay, J. H., Clagett, O. T., and Moersch, H. J.: Experimental Surgery of Thoracic Trachea and Its Bifurcation, Proc. Staff Meet. Mayo Clin. **24**: 555, 1949.

Graham, E. A.: Some Questions About Bronchogenic Carcinoma, New England J. Med. **245**: 389, 1951.

Higginson, J. F.: Block Dissection in Pneumonectomy for Carcinoma, J. THORACIC SURG. **25**: 582, 1953.

Johnson, J., Kirby, C. K., and Blakemore, W. S.: Should We Insist on "Radical Pneumonectomy" as a Routine Procedure in the Treatment of Carcinoma of the Lung? J. THORACIC SURG. **36**: 309, 1958.

Johnston, J. B., and Jones, P. H.: The Treatment of Bronchial Carcinoma by Lobectomy and Sleeve Resection of the Main Bronchus, Thorax **14**: 48, 1959.

Jones, J. C., Robinson, J. L., and Meyer, B. W.: Primary Bronchogenic Carcinoma of Lung, A. M. A. Arch. Surg. **70**: 265, 1955.
Kirklin, J. W., McDonald, J. R., Clagett, O. T., Moersch, H. J., and Gage, R. P.: Bronchogenic Carcinoma: Cell Type and Other Factors Relating to Prognosis, Surg. Gynec. & Obst. **100**: 429, 1955.
Lambert, A.: Radical Surgery in Advanced Cancer of the Lung, New York State J. Med. **55**: 18, 1955.
Lambert, A.: The Importance of Early Diagnosis in the Surgical Treatment of Carcinoma of the Lung, New York State J. Med. **47**: 24, 1947.
Langston, H. T., Laws, J. F., McGrew, E. A., Heindenreich, C., and Slominski, M. D.: The Incidence of Blood Vessel Invasion in Bronchogenic Carcinoma, Surg. Gynec. & Obst. **107**: 704, 1958.
Lindskog, G. E., and Liebow, A. A.: Thoracic Surgery and Related Pathology, New York, 1953, Appleton-Century-Crofts, chapt. 22.
Mathey, J., and Oustrieres, G.: End-to-End Bronchial Anastomosis After Accidental Division of Stem Bronchus, Thorax **6**: 71, 1951.
Meade, R.: Discussion, J. THORACIC SURG. **36**: 324, 1958.
Ochsner, A., Schramel, R. J., Blalock, J., Keeling, J. H., and Haley, J.: Carcinoma of the Lung, A. M. A. Arch. Surg. **74**: 642, 1957.
Ochsner, A.: Cancer of the Lung, Postgrad. Med. **19**: 584, 1956.
Ochsner, A., De Bakey, M. E., DeCamp, P. T., and Ray, C. J.: Bronchogenic Carcinoma; Its Frequency, Diagnosis and Early Treatment, J. A. M. A. **148**: 691, 1952.
Overholt, R. H., and Bougas, J. A.: Common Factors in Lung Cancer Survivors, J. THORACIC SURG. **32**: 508, 1956.
Overholt, R. H., and Bougas, J. A.: Fifty-One Cases of Lung Cancer With Five Year Survival, J. A. M. A. **161**: 961, 1956.
Paulson, D. L.: Survival Rates Following Resection for Bronchogenic Carcinoma, Ann. Surg. **146**: 997, 1957.
Paulson, D. L., and Shaw, R. R.: Preservation of Lung Tissue by Means of Bronchoplastic Procedures, Am. J. Surg. **89**: 347, 1955.
Paulson, D. L.: Survival Rates Following Resection for Bronchogenic Carcinoma, Ann. Surg. **146**: 997, 1957.
Rienhoff, W. F., Jr., King, J. D. B., and Dana, G. W., Jr.: Surgical Treatment of Carcinoma of the Lung, J. A. M. A. **166**: 228, 1958.
Robinson, J. L., Jones, J. C., and Meyer, B. W.: Indications for Lobectomy in the Treatment of Carcinoma of the Lung, J. THORACIC SURG. **32**: 500, 1956.
Sellors, T. H.: *In* Techniques in British Surgery, edited by R. Maingto, London, 1950, W. B. Saunders Co., p. 188.
Shaw, R. R.: Personal communication.
Smith, R. A.: The Results of Raising the Resectability Rate in Operations for Lung Carcinoma, Thorax **12**: 79, 1957.
Thomas, C. P.: Personal communication.
Thompson, V. C.: *In* Diseases of the Chest, edited by G. Marshall, and K. M. A. Perry, London, 1952, Butterworth & Co., Ltd., vol. 2, p. 328.
Tuttle, W.: Discussion of Jackson, Lefkin, Tuttle, and Hampton, J. THORACIC SURG. **18**: 641, 1949.
Watson, W. L.: Radical Surgery for Lung Cancer. Evaluation of the Operation of Radical Pneumonectomy and Five Year End Results, Cancer **9**: 1167, 1956.
Weinberg, J. A.: Identification of Regional Lymph Nodes in the Treatment of Bronchogenic Carcinoma, J. THORACIC SURG. **22**: 517, 1951.

DISCUSSION

(PAPERS BY PAYNE [PAGE 709] AND CHAMBERLAIN [PAGE 727] AND THEIR ASSOCIATES)

DR. FREDERICK R. HARPER, Denver, Colo.—I would like to congratulate Dr. Chamberlain for bringing this to our attention at this time, after Julian Johnson's very discouraging paper of 2 years ago.

I am sure we all have performed extended resections after which the patients survived for some years. The thing that disturbs me is that, for the past few weeks at the Louisville General Hospital, the Pathology Department has been making examinations of the peripheral blood for cancer cells by a technique that has been well described, and in bronchogenic carcinomas the incidence of cancer cells in the circulating peripheral blood is perfectly tremendous. What the significance of these cells is we don't know. We obviously

know from this report and our own work that they do not all survive—that is, these cells—and I hope that more of you will get interested in your institutions and run these sections so that within a very short time we can get some idea of the value or the implications of these carcinoma cells in the peripheral blood.

DR. DONALD L. PAULSON, Dallas, Texas.—My purpose in discussing Dr. Chamberlain's excellent presentation is to emphasize the futility and the possible harm from an overly aggressive attitude in the surgical treatment of bronchogenic carcinoma, and to urge an approach based on, or at least tempered by, the biologic factors of cell type, location of the lesion, and the extent of the lesion.

Certainly one is justified, as emphasized by Dr. Chamberlain, to resect a low-grade epidermoid carcinoma by means of a radical resection in contrast to the small-cell undifferentiated carcinoma, which rapidly disseminates distantly.

This slide shows the operability rates in our series of 1,180 cases from 1946 through 1957, and our experience has revealed a resectability rate, up until 1951, of those patients operated upon of 64 per cent. In the years 1951 through 1955, this rose to 74 per cent for all operations for carcinoma. The operability rate remained close to 50 per cent.

The year 1954 is particularly interesting because we found our resectability rate rising to 83 per cent of all patients operated upon. We felt that this was indicative either of a more aggressive attitude on our part, or that we were dealing with more favorable lesions.

[Slide] A follow-up of these patients operated upon in 1954 revealed no better survival rates than the general average for all the years. Of the 30 patients having pneumonectomy performed in 1954, 3, or 10 per cent, are alive in the fifth year, and of the 34 patients having lobectomy 9, or 26 per cent, are alive. Combining all resections for the year 1954, when our resectability rate was 83 per cent, 12 patients of 64, or 19 per cent, are alive in 5 years, which is the identical figure for all resections done from 1945 to 1956.

[Slide] The figure for all resections is 19 per cent survival for 5 years, lobectomy 23 per cent, and pneumonectomy 15 per cent.

In conclusion, I would like to emphasize the points made by Dr. Chamberlain, that whereas the resectability rate should be maintained at a high level and unwise exploration of a nonresectable carcinoma avoided because of its mortality and lack of benefit to survival time, the tendency on the part of the surgeon to extend the scope of operation and perform palliative resection, knowingly leaving behind carcinoma, is likewise to be deplored. Under these circumstances, and I think Dr. Chamberlain's figures illustrated this, the mortality rate of resections of this type rises close to 20 per cent and survival is no better and, in some instances, not as good as for the inoperable cases.

DR. MICHAEL E. DE BAKEY, Houston, Texas.—I should like to inject one thought here with regard to the evaluation of statistics of this kind, and this concerns again the definition of resectability rates.

Resectability rate of patients operated upon, of course, is one thing. Resectability rate in terms of all cases of carcinoma encountered is still another, and this is important, because obviously unless one relates the patients operated upon to the total number of cases encountered, the resectability rate can vary considerably. It is essential, therefore, to define the resectability rate in these terms, since obviously one can either increase or reduce the resectability rate of all patients operated upon by simply changing the criteria for operation upon all patients encountered.

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—I would like to confine my discussion to the first paper, that of Dr. Moersch and his group.

In studying our group of cases of bronchial adenoma, we have felt that there are well-differentiated factors which, as Dr. Moersch and his group have shown, tend to separate off the cylindromata from the carcinoid group. One of the things which we have studied is the relationship of treatment as one finds the indications in these two groups of tumors.

As they have occurred to us, we have not found a cylindroma that has lent itself to local excision by bronchotomy. On the other hand, we have found 7 patients in whom the carcinoid histology occurred, in which local excision via bronchotomy was a perfectly adequate treatment, and the follow-up in 5 of these has now reached more than 5 years and in 3 of them is approaching 10 years without the evidence of any demonstrable metastasis.

On the other hand, resection of the cylindromatous type with lobectomy has not always been curative, even when we expected cure. In 1 patient recently, 7 years after resection, widespread metastasis of the liver and death from cachexia due to generalized metastasis has already occurred.

It seems that this differentiation in the behavior of these tumors is the real thing on which we can base treatment.

DR. JOHN H. GIBBON, JR., Philadelphia, Pa.—I have a great deal of sympathy with Dr. Chamberlain's point of view about the radical approach to carcinoma of the lung. I don't think my sympathy extends quite as far with regard to palliative operations, but I am not quite sure what Dr. Chamberlain means by this term.

I think resection of the pulmonary veins as they enter the atrium, actually removing a part of the atrial wall, is always indicated if you cannot encompass the tumor otherwise. We excise the tumor when we think that we can remove all gross and visible tumor in the hemithorax.

I have gone so far as to resect the terminal end of the trachea in a right pneumonectomy and anastomosed the left bronchus to the residual trachea. I am sure this is a palliative procedure. This man died from extension 6 months later, and I am not sure how much good we did him. On the other hand, I do think that we should be quite radical in our approach and not back out merely because it is a large tumor.

We have in our group of 5-year survivals 7 patients in whom there was extension into the mediastinum or to structures outside the lung. Two of this group had anaplastic carcinomas.

[Slide] I might just show our experience with cancer of the lung over the past 12 years very briefly here. These patients were operated upon by myself or members of my immediate staff, including surgical residents, so they don't represent quite the type of patient that Dr. Chamberlain referred to in his paper.

We excised the cancer in 40 per cent of all the patients we saw and our 5-year survival rate is 27 per cent of the patients who survived the operations.

I think that Dr. Chamberlain's point is well taken, that this is a disease which is going to kill, and if we can give the patient more years to live by being radical, then I am heartily in favor of it. I would like to say also that we invariably do a really extensive mediastinal node dissection, which I think is an essential part of the operation.

DR. ADRIAN LAMBERT, New York, N. Y.—I should like to discuss the second paper, which I think is a fine presentation, and to say a few words about advanced lung cancer.

Carcinoma of the lung that has invaded the chest wall is extremely important to excise because of the tremendous pain that these patients have when this extension is left in.

This slide is of a cardiac patient in whom there was invasion of the chest wall which was removed at pneumonectomy. He died a year and a half later without being cured of his disease, but his course was not characterized by the excruciating pain which all too frequently leads these patients to suicide.

The second point that I would like to make is that in the patients who have necrosis or hemorrhage from their primary site, that all effort be made to remove the primary tumor, even though one may leave mediastinal glands and obvious malignancy in the mediastinum.

This slide represents a case of that nature in which the lower lobe tumor invaded the left auricle of the heart. This was removed but, because of the patient's general condition, the mediastinal glands were left and appeared enlarged and involved right up to the base of the neck.

[Slide] The next slide shows part of the auricle with the tumor invading it that has been removed. The patient surprised me by not having further extension of his disease until 4 years later, when he developed a node in his neck which was positive for squamous cell carcinoma. Now 6½ years afterward, the patient is about to die of his disease, but in the interim he has been free of his symptoms, which included about a pint of purulent sputum a day and hemorrhage, which had required two transfusions a day for the 2 weeks preceding his operation.

[Slide] The third point I should like to make is that patients who may have blood vessel invasion who later develop a metastasis should have the metastasis considered solitary until proved otherwise. This patient has been commented on before but I show this to you as a follow-up.

This was a 26-year-old girl with adenocarcinoma of the left upper lobe, with no symptoms from her disease, who had been picked up as a contact.

This slide shows the type of adenocarcinoma that she had. We felt we had completely removed it with a left upper lobectomy, taking out about eight mediastinal glands, all of which were negative, until [slide] we saw the malignant cells present in the pulmonary vein. Therefore, we were on the lookout for metastasis so that [slide] 14 months later, when she came in with a cough, and this obvious metastasis in the right upper lobe on the other side, we removed the right upper lobe on that side. The tumor [slide] proved to be the same type as the original tumor.

She then went 6 years and wanted to have a baby. Physiologically, I felt she could withstand pregnancy and advised her to go ahead, which she did. The obstetrician sent her back to me when 4 months pregnant with a lump in the right breast which I thought was probably another metastasis, but in taking it out it proved to be a primary carcinoma of the breast. Radical mastectomy was performed with removal of internal mammary nodes and axillary nodes, all of which were not involved.

It is now 7 years following the primary resection of the breast, 13 years following the primary resection of her carcinoma, and 12 years following the metastasis, and the patient shows no further evidence of malignancy or pulmonary dysfunction.

DR. JULIAN JOHNSON, Philadelphia, Pa.—I certainly appreciate Dr. Chamberlain's paper. I agree with him in principle in the aggressive attitude toward carcinoma of the lung.

Lest there be some misunderstanding regarding our paper last year, we certainly never intended that one should not be aggressive in the removal of a tumor. Our point was that the presence or absence of blood vessel invasion in the tumor is very important in the prognosis. In our practice, if we could remove a tumor grossly at the time of operation we have done so, and that is perhaps attested to by the fact that our 5-year survival rate is 26 per cent, with an over-all 5-year survival of 9 per cent.

The point which we made last year is that so far no one has demonstrated the superiority of a radical operation, such as has not been talked about here but which consists of a widespread mediastinal dissection with removal of the lung and mediastinum en bloc even for an early lesion. Let us not confuse the term. The term "radical pneumonectomy" as proposed by Philip Allison is the widespread dissection for a small lesion, whereas being aggressive in getting a big lesion out is something entirely different. We have felt that one does not need to be obligated to do a wide dissection of the mediastinum, lymph nodes, and contralateral bronchus, take out the pericardium, and divide the vessels to the lung inside the pericardial sac in small lesions, and we ourselves have been doing more and more lobectomies for the small lesion. We have our highest 5-year survival rate in that group.

But as far as taking the big tumors out, we are not relieved of our obligation there in any way by these data which we gave on 5-year survival. We don't know at the time of operation whether there is blood vessel invasion or not, and if we have a huge tumor without blood vessel invasion and do not take it out, we have done a disservice to that patient.

This was attested to by 1 such patient in our series. I found, to my surprise, after I had terminated the operation and sent him home to die, that 5 years later he was still alive.

We went back and reviewed his tumor and he did not have blood vessel invasion in his tumor. I reviewed our operative note and found that he had had invasion of the right atrium which was the reason we stopped. If we had been a little more aggressive perhaps we could have got this out and the man could have been saved. Although he is obviously going to die of his tumor, he is still alive almost 6 years later with no resection at all. He had had no x-ray treatment, no nitrogen mustard, or specific therapy of any kind.

DR. ROBERT J. JENSIK, Chicago, Ill.—I came to this meeting anticipating a good report by Dr. Chamberlain and he certainly didn't disappoint me, and I am sure he didn't disappoint the rest of us.

I would like to sympathize with his approach to this problem and give him every support that is possible, and mention a few of our figures along this line. We have had much the same philosophy and, in comparing 5-year periods of 1949 to 1953 and 1953 to 1958, we find that we have increased the resectability rate in the first group of 61 per cent to 80.5 per cent in the second group.

In the second group there were 282 thoracotomies and resection was accomplished in 227 patients.

These patients represent private practice material entirely and we do have the opportunity of comparing clinic material. Notoriously, clinic material does not give the same ideal type of patient, because in many of them the disease is so far-advanced our resectability figures are as low as 60 per cent in that group.

I wanted to ask Dr. Chamberlain about the problem of hoarseness, whether he uses that as a contraindication to a thoracotomy. We have felt that even if the patient demonstrated a paralyzed cord he was still entitled to an operative chance and in about 25 patients have accomplished resection in 10 of these. We have not had a single 5-year survival in a patient in whom hoarseness was a problem, the longest survival being 3 years.

DR. LAURENCE K. GROVES, Cleveland, Ohio.—I should like to point out another area where guarded aggressiveness may be indicated.

[Slide] This is not, certainly, a typical Pancoast's syndrome, so-called, but this patient's chief complaint was pain in the region of the right shoulder. He had a lobectomy and the region of the pleural apex, where there was frank tumor left behind, was infiltrated with radon seeds. The date isn't all on there; it is December, 1953.

I had a letter from him last week. He is fine. When he started coming back about 3 years later we got interested in trying this on some other patients. None of them has done as well as he has, but is has been impressive to us that if there isn't frank brachial plexus involvement it is surprising that even the next day, after you have avulsed the tumor off of the apex and put radon seeds in, there seems to be a peculiar relief of pain, gratifying palliation, and much more comfort.

DR. AARON HIMMELSTEIN, New York, N. Y.—I would like to comment about one point that Dr. Chamberlain has raised, namely, sleeve resection. The particular comment is to its value in the preservation of function.

As you see, this slide is the preoperative x-ray film of a patient with a lesion in the upper lobe. It was seen by bronchoscopy but could not be biopsied.

[Slide] Here is the x-ray about a month postoperative.

[Slide] Here is an x-ray more than a year postoperative and he is alive and well.

[Slide] This color photograph of the specimen shows the type of resection taking the sleeve of main bronchus.

[Slide] This shows the bronchus open.

[Slide] This is the last one, which is the point of the discussion, and shows the bronchospirometry results after a year. The slope of these curves shows the oxygen consumption on each side. The resection was on the right side, with oxygen consumption on

this side being 33 per cent of the total. These cases have interested us particularly because they also offer one the opportunity to study a lung which has been largely denervated, since the pulmonary artery is stripped of its neural sheath and the only structure which is relatively undivided or untouched is the inferior pulmonary vein.

The relationship of circulation to ventilation in these lungs also is of great interest.

DR. JEROME R. HEAD, Chicago, Ill.—I would like to say a few words about Dr. Payne's excellent paper. It seems to me it is important in that it shows definitely the malignancy of the cylindromas and the mucoepitheliomas, but also because, by implication, it implied the nonmalignancy of the carcinoid tumors or the simple adenomas.

I feel that simple adenoma is never an indication for lung resection unless the lung has already been so damaged by bronchial destruction that it is past redemption. I agree with Dr. Goldman that, in these cases, a bronchotomy is the procedure of choice if the lesion cannot be removed through the bronchoscope.

I would like to mention one unusual procedure which we used in a case of this kind in a woman with a right upper lobe atelectasis. On bronchoscopy we found a simple adenoma protruding from the right upper lobe bronchus. At that time we were using flammable anesthetics and could not use diathermy. We did a bronchotomy and found that both the upper lobe bronchus and part of the main bronchus were involved with the tumor. We closed off the main bronchus and packed the intermediary bronchus; we then took a uterine curet and curetted the upper lobe bronchus. Bleeding, as is usual in these cases, was profuse and was uncontrollable at the time of operation, so we packed the upper lobe bronchus with gauze and left it in place, but attached a long string to it, which we brought out through the mouth. We left the pack in place for 48 hours and then withdrew it through the mouth.

DR. PAYNE (Closing).—I wish to thank Dr. Goldman and Dr. Head for their comments. Although we consider carcinoid bronchial adenomas, muco-epidermoid tumors, and cylindromas of the bronchus to be distinct and separate clinical and pathologic entities, we also regard them as malignant. As has been indicated in previous publications from our institution, metastasis is a feature of about 10 per cent of bronchial carcinoid tumors. It is our feeling at present that surgical resection is the preferred form of treatment for carcinoid tumors as well as for the other two tumors under discussion.

We should like to emphasize two additional points. First, although the cylindroma may appear to be inoperable by ordinary criteria, very excellent palliation can be obtained by resection, even when tumor must be left behind. The low incidence of vascular invasion probably accounts for the better prognosis associated with this tumor as compared to the prognosis for the usual bronchogenic carcinomas.

Second, although muco-epidermoid tumors and many of the carcinoid tumors are of a low order of malignancy and may be removed adequately by sleeve resection of the bronchus or by more conservative means, the pulmonary sequelae of bronchial obstruction may require more radical resection.

DR. CHAMBERLAIN (Closing).—I would like also to thank the discussers who complimented me by discussing this paper.

I might answer Dr. Jensik's question first by saying that his experience and mine are essentially the same. We did not go into the indications in this 15-minute presentation, but hoarseness is usually a contraindication unless a palliative operation is planned. Of course, on many occasions I have had to perform a biopsy on these people because the x-ray therapist will not treat them unless we have a histologic diagnosis. In some cases, when hoarseness is not the major symptom, I have removed the recurrent laryngeal nerve, as I am sure you have, hoping to cure the patient.

I believe I can answer the other discussers together: Dr. Paulson and Dr. Gibbon. It is interesting to me that they were the ones who called your attention to a cautious approach, since I have recently gone over their published reports in detail. Their resectability rates

are higher than the average in this country, so it is not surprising that they should warn you to be cautious. Dr. Gibbon's paper points out that approximately 50 per cent of his patients have had pericardiectomies, and Dr. Paulson's and Dr. Shaw's resection rate is up in the 70's, as he pointed out to you.

I understood Dr. Johnson's presentation in Boston exactly as he recorded it to you today. I am sure that he is as anxious to resect these lesions as any of us.

If all of us were to be completely honest with ourselves, we must admit that our ability to resect advanced cases of carcinoma *now* is much greater than it was the day we finished our residencies, and my plea before this Society is to insist in our teaching environs that when we send these young men out into the world that they have with them the technical scope and the technical ability to do an "extended resection" should they encounter the need for it.

Now I should like to say a word or two in regard to those cases in which we are forced to leave cancer behind. At the beginning of an operation a surgeon does not always know that he is going to leave cancer behind. He may think, "Well, this lesion is completely resectable," and go ahead with the intention of resecting it, even though it is necessary to take the pericardium and the atrial wall, etc. However, when the operation is almost completed, it is at that time that he discovers that there is still inaccessible carcinoma. I do not like to leave carcinoma behind, but on occasions there is no other solution.

Sir Clement Price-Thomas kindly sent me a copy of his Tudor Edwards Memorial Lecture just prior to presenting it before the British Thoracic Society in May, 1959. It is of interest that the essence of his presentation was a plea for extended efforts to resect carcinoma of the lung. In 306 patients operated upon prior to 1951, his resectability rate was 60 per cent. In 268 patients operated upon since 1951, his resectability rate was 75 per cent. His published report will show that extended operations for carcinoma of the lung may be performed with encouraging results.

For those of you who may return to your respective environs with a more aggressive approach, I would like to read this line to you again: "It is not our intent to encourage unguarded aggressiveness in the modern thoracic surgeon."

INSTANTANEOUS BLOOD LOSS DETERMINATION DURING THORACIC SURGERY

Robert Klopstock, M.D., Harry H. LeVeen, M.D.** (by invitation), and P. I. Levitan, M.D.*** (by invitation), Brooklyn, N. Y.*

THE MAGNITUDE and duration of present-day surgery, in general, and of thoracic and cardiovascular surgery, in particular, would not be possible without adequate blood replacement. As magnitude and duration of surgical procedures expand, the volume of operative blood loss increases, necessitating replacement of larger and larger amounts of blood. Anatomic structures dealt with in thoracic and cardiovascular surgery carry the risk of brisk hemorrhage on occasions and the demand of speedy restoration of the circulating blood volume. How then may one best maintain an adequate blood volume and at the same time prevent undertransfusion or circulatory overloading?

The work of Ebert and Stead[1] clearly indicates that during the course of blood loss in normal donors there is no gradual decrease in blood pressure and pulse rate. These authors state, "the onset of collapse was sudden. Until it occurred, the subject had no complaints. In three subjects collapse developed from 1 to 4 minutes after venous suction was completed. The compensatory mechanism appeared to break down suddenly even though no more blood was being removed and the position of the subject had not been changed." This fact is well illustrated by a diagram which has been adapted from Barcroft and others[2] (Fig. 1) to demonstrate the changes in vital signs during the brisk hemorrhage in a normal donor. The blood pressure which starts in the neighborhood of 120 does not fall, the pulse rate rises but little, and yet the cardiac output continuously diminishes. It is only when compensation fails that the blood pressure drops suddenly. This shows that the blood pressure and the pulse rate cannot be relied upon as an index of adequate replacement. Neither the rise in pulse rate nor the fall in systolic pressure makes its appearance until the volume of blood loss is greater than can be compensated for by vasoconstriction. This usually does not occur until after 800 to 1,500 c.c. of blood is lost, if the

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patient is a well nourished and hydrated adult. Although venous pressure is of some value in determining the extent of blood loss, it becomes valueless during rapid bleeding and rapid replacement. During severe hemorrhage the sudden replacement of blood may fail to bring about a recovery in the hemodynamic alterations even when replacement has been of the proper amount. There have been various explanations for this failure of transfusion to prevent the disturbances in hemodynamics, some of which have been ascribed to the high citrate concentration of bank blood. The citrate in blood enhances the action of potassium by removing its ionic antagonist, the calcium. The potassium concentration of bank blood rises to 25 mEq. per liter by the end of 2 weeks. The rapid infusion of this bank blood may induce cardiac arrest or a state not unlike that of forward circulatory failure. It is under these circumstances that

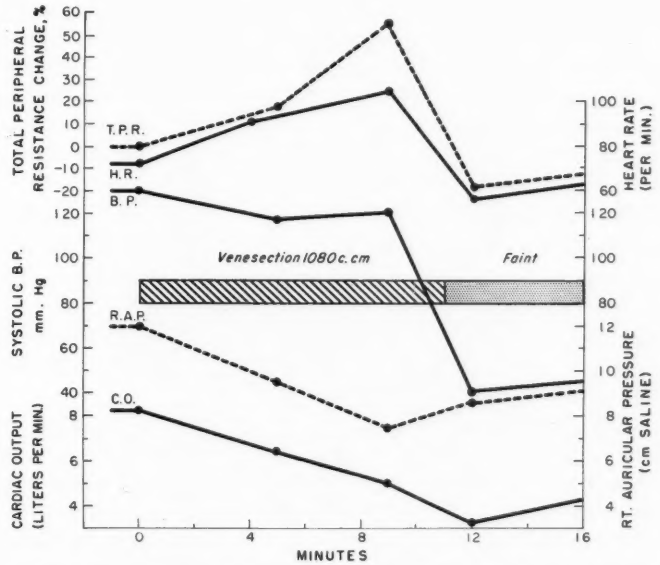

Fig. 1.—The course of events during rapid venous section of a normal donor is charted. Blood pressure (*B.P.*) falls precipitously from 120 to 40 mm. Hg. The heart rate (*H.R.*) does not rise above 100. The cardiac output (*C.O.*) falls continuously. The total peripheral resistance (*T.P.R.*) rises until decompensation occurs. Right auricular pressure (*R.A.P.*) falls continuously during the bleeding.

venous pressure will fail to differentiate between overtransfusion and forward circulatory failure induced by high potassium concentration of bank blood. It is, therefore, obvious that if accidents are to be avoided during surgery entailing massive blood loss that the amount of loss must be continuously measured during the operative procedure and exact replacement made for the amount which is lost.

Since the first study of blood loss during some of the most common operations was reported by Gatch and Little[3] in 1924, many investigators have made extensive measurements of operative blood loss in the various fields of surgery

and concluded that volume lost was always greater than the surgeon estimated and that the circulatory volume may be decreased considerably before clinical shock is noted.

Great credit should go to Wangensteen[4] for having suggested (1942) the relatively simple and accurate method for determining operative blood loss *as it occurs* by weighing the dry sponges before and after their use and thus calculating the blood lost during surgery.

In the field of thoracic surgery, White and Buxton,[5] then Miller, Gibbon, and Allbritten,[6] made very valuable studies of blood loss during the various major thoracic procedures. Gibbon[6] and his associates pointed out that they were unable to estimate the amount of blood which had been lost and the amount which should be replaced by observing the vital circulatory signs.

Operative blood loss has previously been determined by both the gravimetric and the colorimetric methods. Blood volume studies were done by the dye dilution method, according to Gibson and Evans,[9] and by radioactive tagging of blood components, and the blood loss has been calculated in these ways. However valuable these methods are, they are not very suitable for the routine and everyday evaluation of blood loss in the operating room when extensive procedures of long duration are carried out.

The possibility of using *the phenomenon of electrical conductivity* in the determination of operative blood loss has been investigated by Harry H. LeVeen.[7]

The conductivity method is based on the fact that blood is a solution of almost constant electrolyte composition. Even in "low salt syndrome" and other more serious disease states, the total sodium does not vary by more than 10 to 20 c.c. mEq. per liter. The extent of blood loss can therefore be assessed by measuring the quantity of lost electrolytes.

The electrical resistance of a solution decreases with increasing electrolyte concentration. More concentrated solutions of electrolyte will allow for greater passage of current according to Ohm's law. If blood is added to water in varying concentrations, a direct proportional relationship between concentration of blood and conductance results. Since small changes in conductance can be accurately measured with a Wheatstone bridge, the change in conductance produced by the the addition of blood to known volume of water can be determined precisely. With a suitable Wheatstone bridge and proper temperature compensation, determinations are accurate to 0.5 per cent.

LeVeen,[7] utilizing the discussed principles, carried out extensive preliminary experiments and devised an apparatus, the blood loss monitor, which calculates and instantaneously registers all blood delivered into it.

DESCRIPTION OF THE BLOOD LOSS MONITOR

The apparatus consists of a large stainless steel tub with an agitator which continuously extracts the electrolytes from the sponges and clots. A suction pump transfers blood from the operating table directly into the tub (Fig. 2). This procedure makes possible the addition of blood loss taken up via suction as well as from bloody sponges. Since conductivity increases with increasing temperature of the solution, automatic temperature compensation was

accomplished by introducing a thermistor into one arm of the conductivity bridge. The thermistor assumes the temperature of the water in the reservior and changes the resistance of one arm in the bridge to compensate for the decreasing resistance of the solution at increased temperatures. It was desirable to have a direct reading instrument which would obviate the difficulties of manually balancing the conductivity bridge. Hence, a servomechanism was coupled with the conductivity bridge. The servomotor continuously brings the bridge to the null balance point. Instantaneous and continuous detection of conductivity changes are made. The conductivity bridge and cell must be specially constructed to operate on low voltages in order to minimize polarization and, at the same time, maintain its sensitivity. All components of the machine are constructed to meet explosion proof requirements

Fig. 2.—A photograph of the blood loss monitor is shown in simulated use. The bloody sponges are dropped in the open tub. The suction is connected to the side of the apparatus. The dial face can be seen.

for use in an operating room. The bridge is calibrated in cubic centimeters of blood rather than in solution conductance (the unit of electric conductivity). Blood loss is measured in increments of 100 c.c. up to 8,000 c.c. and with possible estimate of 25 to 50 c.c. within the increment. There is also an arrangement to measure small volume of blood loss from 0 to 800 c.c. to be used in surgery of children. This dial can be read with an accuracy to 20 c.c. and estimated to 10 c.c. within the increment (Fig. 3). This range can be put into operation by turning the proper switch. A centrifugal pump empties the tub following usage and the

tub can be rapidly emptied into a regular sink and refilled so that losses greater than 8,000 c.c. can be measured. The machine is cleaned with a chlorinated detergent following use and rinsed with tap water. A stainless steel measure is used to measure the initial quantity of tap water placed in the tub. Fixed resistances with push buttons are included so that the

Fig. 3.—The dial face together with the range switch is illustrated in detail. When the switch is in the "adult" position readings are made on the outer circular scale. When the switch is in the "child" position the inner circular scale is used for reading.

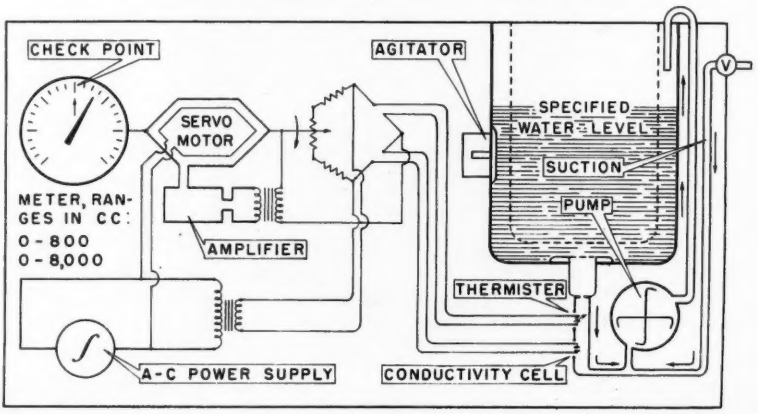

Fig. 4.—Schematic diagram of the automatic blood loss meter. A measured volume of distilled water (25,000 c.c.) is added to the tub. An agitator extracts electrolytes from the sponges, lap pads, and clots. A suction apparatus transfers blood from the operative site to the tub. Automatic temperature compensation is accomplished with a thermistor which forms one arm of this bridge. The conductivity cell is also shown. A servomotor continuously keeps the bridge at the null balance point. The dial of the meter is calibrated directly in cubic centimeters of blood. Sponges and laparotomy pads are held in a stainless steel basket indicated by broken line inside container.

calibration of the machine can be pre-tested before use. A null balance of 0 c.c. of blood is automatically accomplished by using tap water of low conductivity or distilled water. Correction for the initial conductivity of the water is then unnecessary. A schematic drawing of the instrument is shown in Fig. 4.

Remarks.—It is evident that sodium chloride solutions cannot be used for irrigation purposes or to moisten sponges and pads unless a definite measure of the amount of sodium chloride used is made and subtracted from the final total of indicated blood loss. We use a solution of 2.5 per cent glucose for moistening laparotomy pads for irrigation. Since glucose is a nonelectrolyte, it does not interfere with the readings.

OBSERVATIONS

The blood loss monitor was used in 62 patients who underwent major thoracic and cardiovascular surgery.* In Table I, the observed minimum and maximum blood loss values are tabulated for each type of operation and the calculated averages are noted. These are approximately equal to those reported by other authors, notably by Miller, Gibbon, and Allbritten.[6] Blood loss sustained up to the opening of the pleural cavity ranged 150 to 750 c.c. with an average of 364 c.c. It should be stressed that there was no need in any case for additional blood replacement after completion of the surgical procedure if blood replacement was made according to the indication on the monitor. In all cases there was less than 1.2 Gm. change in the hemoglobin value on the first to the fourth day as compared with preoperative values. Thornton and Adams[8] pointed out that the amount of hemoglobin tends to drop postoperatively in those cases where blood replacement has been inadequate. This postoperative drop in hemoglobin value described by Thornton and Adams did not occur in our cases in which the blood replacement was carried out under the guidance of the blood loss monitor.

There were only 2 cases (No. 1 and No. 2) in which shock occurred during surgery, and these observations are described fully because of their study value.

COMMENT ON ILLUSTRATIVE CASES

In this instance it was decided to compare the accuracy of the anesthetist's clinical estimate to blood loss registered by the monitor. Therefore, the "dial indication" of blood lost was unknown to him at the time vital signs had changed.

CASE 1.—*Lobectomy for Carcinoma With Chest Wall Involvement.*—

The patient was a 39-year-old man with a round tumor in the right upper lobe. On exploration of a carcinoma, it appeared that it was anatomically resectable by means of an upper lobe resection, even though the tumor-bearing part of the lobe was densely adherent to the parietal pleura in a circumscribed area. In addition, the entire upper lobe was attached by vascular adhesions to the parietal pleura. Therefore an extrapleural dissection was carried out. This patient exhibited an unusual degree of oozing over a prolonged period of time. During the procedure, the degree of blood loss was not appreciated by the anesthetist until the vital signs changed. At this time 3,500 c.c. of blood loss was registered *while* only 2,000 c.c. had been replaced (Fig. 5). The known deficit was corrected gradually and the patient's condition returned to normal. Total blood loss and replacement for the operation was 7,000 c.c. In the postoperative period, no additional blood replacement was necessary.

This observation illustrates the wide discrepancy between clinical impression and accurate measurement of blood loss. It is in such cases, when the blood loss is small but continuous, that undertransfusion is common.

*See Addendum.

TABLE I. OPERATIVE BLOOD LOSS MEASURED BY MONITOR

TYPE OF SURGERY	NO. OF CASES	MINIMUM	MAXIMUM	AVERAGE		SHOCK		ADDIT. REPLACEMENT 48 HR. POSTOP.	REMARKS
				AUTHOR	MILLER, GIBBON, ALLBRITTEN	DURING OPERATION	48 HOURS POSTOP.		
Miscellaneous chest wall procedures	4	450	3,500	2,200		–	–	–	
Exploratory thoracotomy	9	150	3,000	1,006	1,074	–	–	–	
Pulmonary wedge resection	3	450	950	767		–	–	–	
Segmental resection	14	1,150	2,500	1,812		–	–	–	
Lobectomy	12	950	2,850	1,724	1,778	–	–	–	
Lobectomy (cancer, chest wall)	1	7,000				1*	–	–	*Short duration (considered in detail)
Lobectomy + segment	4	1,500	3,050	2,252		–	–	–	
Pneumonectomy	4	1,650	2,250	1,825	1,915	–	–	–	
Decortication	1		1,400			–	–	–	
Mediastinal tumor	1		1,050			–	–	–	
Mitral commissurotomy	4	950	2,500	1,513		1†	–	–	†Short duration (considered in detail)
Esophageal surgery	3	1,700	2,650	2,105	1,876	–	–	–	
Pericardectomy	1		1,050			–	–	–	
Resection of aneurysm of thoracic aorta with hypothermia	1	6,200				–	–	–	(considered in detail)
Total	62								
On entering pleural cavity	58	150	750	364					

Less than 1.2 Gm. change in hemoglobin for all cases, 1 to 4 days postoperative as compared to preoperative values.
*Case 1.
†Case 2.

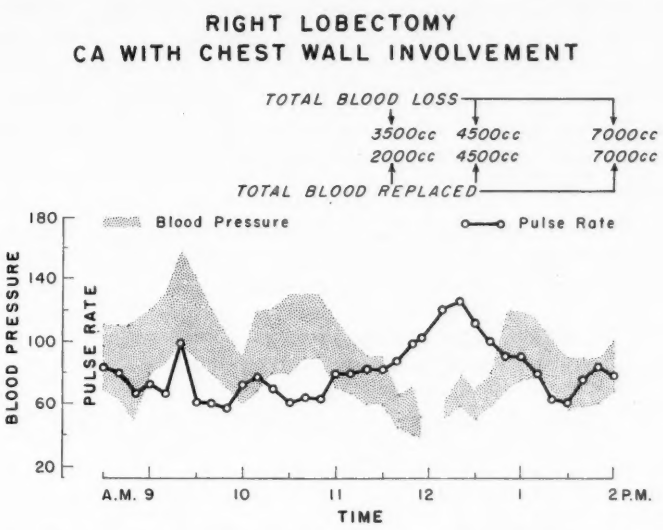

Fig. 5.—Illustrates not only the ease of management of a patient who during surgery had gradual blood loss of 7,000 c.c. with change in the hemodynamics but also the facility with which known large deficits can be readily corrected.

CASE 2.—*Mitral Commissurotomy.*—

The patient was a 37-year-old white man with mitral stenosis. During the otherwise uneventful procedure there was a sudden loss of 1,200 c.c. of blood following tear of the left atrium and superior pulmonary vein. Blood pressure was unobtainable for a short period of time, but returned to normal after replacement of the indicated volume. The patient did well following surgery and no further blood replacement was needed (Fig. 6).

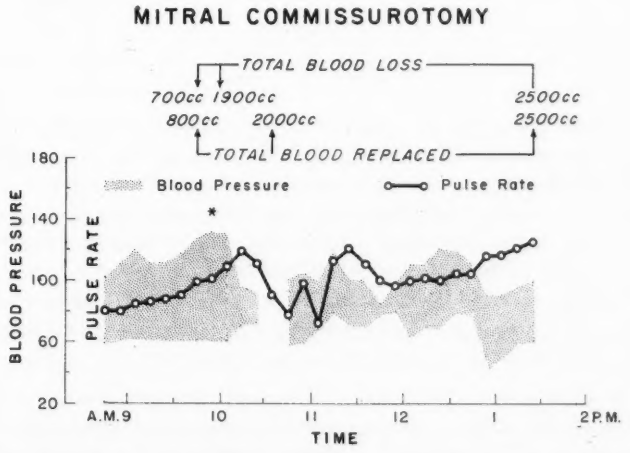

Fig. 6.—Brisk blood loss precipitating shock during cardiac surgery is easily managed by monitoring and replacing the blood loss as it occurs.

When blood loss is sudden and massive one may "get lost" in the management of replacement if it is based on clinical estimate. It is in such cases that overtransfusion, most commonly, and cardiac arrest, occasionally, are induced.

The train of events substantiates also the experimental data on normal donors which indicate that vital signs do not change until considerable blood has been lost and only then they change suddenly.

Hypothermia.—

The increasing use of hypothermia poses problems for both surgeon and anesthesiologist. Not only does hypothermia render the vital signs valueless, but it also enhances the tendency toward cardiac arrest. During hypothermia,

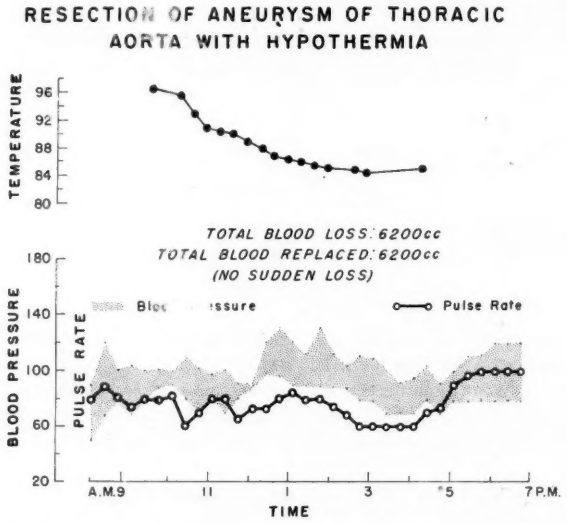

Fig. 7.—Monitored blood loss and replacement during 10-hour hypothermic vascular surgery is graphically shown.

patients tolerate blood loss very poorly and the blood volume is restored very slowly after hemorrhage. This is partly brought about by a fall in the osmotic pressure of the plasma protein during hypothermia. Under these circumstances fluid cannot be rapidly mobilized from the tissue spaces. The instantaneous determination of blood loss was of considerable aid not only in thoracic surgery, but in all surgery performed with hypothermia.

The following case report illustrates the usefulness of monitored blood loss in cardiovascular surgery carried out under hypothermia with necessarily hypotensive blood pressure levels and altered pulse rate.

CASE 3.—*Excision and Replacement of Descending Thoracic Aorta.—*

The patient was a 42-year-old white man with a large fusiform aneurysm of the descending thoracic aorta. At exploration the dilatation was found to extend from just distal to the left subclavian artery to a point just above the diaphragm. Under hypothermia a

Teflon prosthesis was used to bypass the aneurysm which was then resected; the prosthesis was left as a permanent bypass. There was no dramatic blood loss during the 10-hour procedure, but after replacing the volumes of blood indicated by the monitor, it was found that 6,200 c.c. of blood had been lost by continual ooze. The patient had an uneventful postoperative course and no further blood replacement was necessary (Fig. 7).

In the properly monitered case, blood deficit and overtransfusion do not occur if loss and replacement are checked periodically during the course of prolonged surgery. Hourly check should be considered the minimum interval during the uneventful progress of an operation. A typical chart of monitored blood loss is shown in Fig. 8.

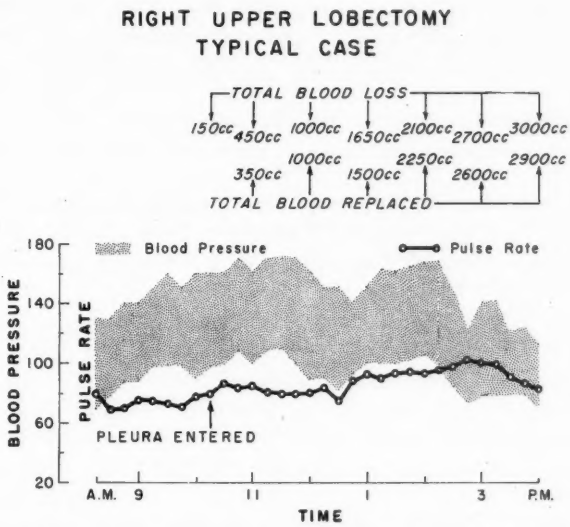

Fig. 8.—The matching of blood replacement to blood loss is shown in a typical intrathoracic surgical procedure.

GENERAL COMMENTS

In the foregoing, data are presented to document the value of an apparatus, the blood loss monitor, for accurate determination of blood loss during surgery as it occurs. The construction and function of the apparatus is based on the fact that the blood is a solution of almost constant electrolyte composition. The extent of the blood loss is assessed by measuring the quantity of lost electrolytes.

Since the characteristics of blood as an electrolyte are utilized as a measurement of blood, all the factors affecting conductivity in blood must be reckoned with.

Nonelectrolyte in high enough concentration will effect a reduction in conductivity. The usual concentration of glucose and nonprotein nitrogen in blood do not produce a perceptible change. Serum protein and hemoglobin do influence conductivity in an ideal way. For each gram per cent deficit in plasma

protein, conductivity increases approximately 2 per cent. A blood loss of 100 c.c. in a patient with a gram per cent deficit in plasma protein would appear as a loss of 102 c.c. This would cause the patient to be overtransfused by 2 per cent. A change in hematocrit from 50 to 5 will register approximately 20 per cent increase in loss. An anemia of 50 per cent would bring about an over-replacement of blood loss by an amount slightly in excess of 10 per cent. The method makes moderate corrections for severe hypoproteinemia and anemia.

Open Heart Surgery.—We have not yet had the opportunity to use the blood loss monitor in open heart surgery, but it seems apparent that such an apparatus could be of inestimable value.

The accurate determination of the blood lost before the perfusion begins could be accomplished with ease. During perfusion all blood lost from the oxygenator-patient-circuit must be accurately known so that correct volume of blood may be maintained in the patient and the perfusion terminated with the patient in normovolemic state. In infants, of course, this assumes even greater importance since small amounts of blood represent larger proportions of total blood volume.

Rapid losses of large quantities of blood during cannulation as occurs on occasion, or accidental injury to major vessels in the preparation for perfusion, may prejudice the outcome if fast and accurate replacement is not carried out prior to the total body bypass.

Compensated Hypovolemia.—In considering the efforts to safeguard the surgical patient against the hemodynamic changes brought about by over- or undertransfusion, attention should be directed to the possible danger inherent in compensated hypovolemia. Recent experiment of ours had shown that potassium excretion is impaired in compensated hypovolemia even though blood pressure and pulse rates remain unchanged. During compensated hypovolemia in animals, serum potassium levels will rise with infusions of hyperkalemic blood (bank blood). This rise does not occur in normovolemic animals. The rise of serum potassium exposes the animals to the possibility of cardiac arrest. These experimental observations confirm the effort that blood loss be replaced quantitatively as it is lost.

In Conclusion.—The feeling is expressed that the instantaneous and continuous determination of operative blood loss gives the surgeon the assurance that the patient's hemodynamics will not undergo sudden collapse and that the patient will not be exposed to biochemical changes making him susceptible to cardiac arrest because an exact replacement is made as blood is lost. The maintenance of hemodynamic equilibrium enhances greatly the execution of the purpose of surgery and safeguards the patient from measures which are necessitated under the duress of hemorrhage.

SUMMARY

Instantaneous and continuous determination of operative blood loss was carried out in 62 major thoracic procedures utilizing an apparatus, the blood loss monitor, based on the principle of electrical conductivity of the blood.

Results are tabulated.

Pathophysiology of response to blood loss has been discussed.

The method proved to be simple, reliable, accurate, and extremely useful.

ADDENDUM

Since this report was submitted, the blood loss monitor was used in 57 additional major thoracic and cardiac operations. In none of the patients did shock occur during or after the completion of surgery, and in no instance was it necessary to use additional blood replacement beyond the amount indicated by the monitor during the surgical procedure.

REFERENCES

1. Ebert, R. V., Stead, E. A., and Gibson, J. G.: Response of Normal Subjects to Acute Blood Loss, Arch. Int. Med. **68**: 578, 1941.
2. Barcroft, H., Edholm, O. G., McMichael, J., and Sharpey-Schafer, E. P.: Post Haemorrhagic Fainting, Lancet **1**: 94, 1944.
3. Gatch, W. D., and Little, W. D.: Amount of Blood Loss During Some of the More Common Operations, J. A. M. A. **83**: 1075, 1924.
4. Wangensteen, O. H.: The Controlled Administration of Fluid to Surgical Patients Including Description of Gravimetric Methods of Determining Status of Hydration and Blood Loss During Operation, Minnesota Med. **25**: 783-789, 1942.
5. White, M. L., and Buxton, R. W.: Blood Loss in Thoracic Operations, J. THORACIC SURG. **12**: 198, 1942.
6. Miller, B. J., Gibbon, J. H., Jr., and Allbritten, F. F., Jr.: Blood Volume and Extracellular Fluid Changes During Thoracic Operations, J. THORACIC SURG. **18**: 605, 1949.
7. LeVeen, H. H., and Rubricius, J. L.: Continuous, Automatic, Electronic Determinations of Operative Blood Loss, Surg. Gynec. & Obst. **106**: 368, 1958.
8. Thornton, T. F., Adams, W. E., Bryant, J. E., and Carlton, L. M.: The Use of Whole Blood Transfusions in Resections of the Lung, J. THORACIC SURG. **14**: 176, 1945.
9. Gibson, J. G., and Evans, W. A.: Clinical Studies of Blood Volume, J. Clin. Invest. **16**: 301, 1937.

DISCUSSION

DR. KLOPSTOCK.—In closing, I would like to show a short motion picture to demonstrate the blood loss monitor in simulated action. Here you see the apparatus with the dial face registering blood loss up to 8,000 c.c. Before using, 0 point is being tested. Now 1,000 c.c. of blood is delivered into the tub, watching the dial you see that it instantly registers 1,000 c.c. Then the content of the suction bottle, containing 450 c.c. of blood, is suctioned into the tub and you can notice that the registered total is now 1,450 c.c. Bloody sponges and pads are now dropped ino the tub, registering an additional 200 c.c.—altogether 1,650 c.c. of blood loss. Excessive foam formation in the tub solution is prevented by addition of an antifoam agent. Thank you.

USE OF A MECHANICAL RESPIRATOR IN THE MANAGEMENT OF RESPIRATORY INSUFFICIENCY FOLLOWING TRAUMA OR OPERATION FOR CARDIAC OR PULMONARY DISEASE

Frank C. Spencer, M.D., Donald W. Benson, M.D. (by invitation), W. C. Liu, M.D. (by invitation), and Henry T. Bahnson, M.D., Baltimore, Md.*

THE USE of a mechanical respirator to maintain patients with severe pulmonary insufficiency through the critical period after pulmonary resection was first described by Björk and Engström in 1955.[1] Subsequent experience has shown the validity of their premise, that a satisfactory recovery is possible if the patient can be helped to survive the critical early period after operation when respiratory function is poor because of trauma to the lung, increased secretions, pain, and fatigue. Avery, Mörch, and Benson[2] have used a respirator to relieve respiratory insufficiency resulting from a flail chest after crushing injuries, the respirator being used in some instances until the rib cage became stable enough to prevent embarrassing paradoxical breathing. In 1957, Björk and Engström[3] had used the respirator in 61 patients, including 3 in pulmonary edema, and mentioned in an addendum the use on patients following cardiopulmonary bypass and open heart surgery.

We wish to emphasize the value of a respirator after open heart surgery to carry some patients through the early postoperative period when survival is not possible without this supportive measure. In the cases to be reported, respiratory insufficiency followed trauma in 1 case, pulmonary surgery in 2, and cardiopulmonary bypass and open correction of cardiac defects in 7.

THE RESPIRATOR

The Mörch respirator used in this group of patients consists of a piston, the stroke of which can be varied from 0 to 3.2 liters by changing the radius on a crank. The crank in turn is driven by an electric motor with a variable speed reduction unit which allows regulation of the rate from 0 to 40 strokes per minute. The stroke volume and cycling rate are the only adjustable factors. Air is pulled into a chamber through a one-way valve by the backward movement of the piston and is then pushed out by the forward movement through

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*Dr. Spencer is a John and Mary R. Markle Scholar.

a humidifier to a valve placed as close to the patient as possible. During the inspiratory stroke, air is forced through this valve to the patient. When the respirator is filling, the same valve allows passive exhalation by the patient to the atmosphere and at the same time stops aspiration of the patient's expired air into the apparatus. The respirator has been previously described,[2] and a diagram is shown in Fig. 1.

This apparatus has the advantages of a large stroke volume and simplicity of management. The large and certain stroke volume allows one to hyperventilate almost any patient sufficiently to produce slight hypocapnia and alkalosis and thus control the ventilation without resistance from the patient.

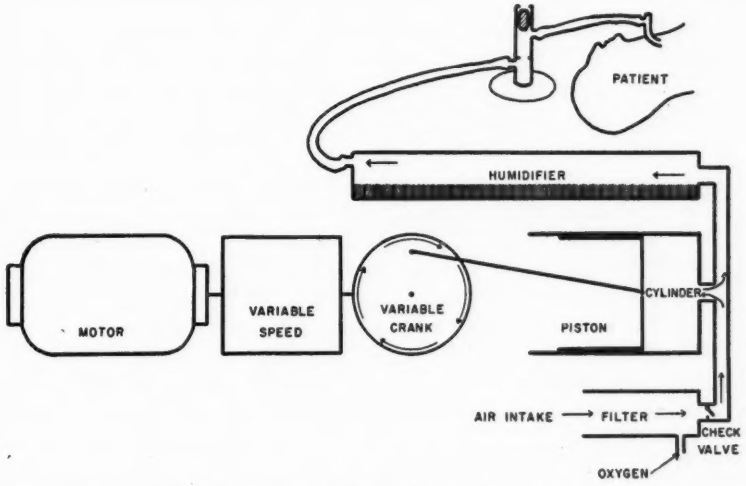

Fig. 1.—A diagram of the construction of the respirator is shown. The valve is placed as close to the tracheotomy tube as possible.

In addition, a loose tracheotomy tube can be used with the large stroke so that pressure necrosis does not occur and secretions can be blown up around the tube. A precise regulation of stroke volume is not needed as excess volume is simply blown past the loose tube. In practice, stroke volume and rate are initially increased to produce apnea and then reduced to a point just above that at which the patient makes respiratory effort. At this level of ventilation, arterial pH is usually mildly alkalotic (pH 7.45 to 7.60). Rate and stroke are subsequently altered to match the patient's demands and to maintain mild hypocapnia and alkalosis.

Humidification to prevent drying of the tracheal mucosa is an important consideration with the large volume of gas used. The respirator is equipped with a humidifier but this was supplemented by instilling 5 ml. of sterile saline into the tracheotomy tube every 30 minutes and, in some instances, by connecting an intravenous saline drip to the tube near the patient and running this slowly. Inadequate humidification was probably the cause of the single death reported in this group of patients (Case 4, Table I). She ultimately died with

TABLE I

CASE NO.	NAME, AGE, SEX	DIAGNOSIS	HISTORY OF: DYSPNEA	HISTORY OF: CONGESTIVE HEART FAILURE	PULMONARY ARTERY PRESSURE (mm. Hg)	OPERATION	INDICATIONS FOR USE OF RESPIRATOR	POST-OPERATIVE DAY RESPIRATOR BEGUN	DAYS RESPIRATOR USED	RESULT
Case 1	E. P. 34, F	Atrial septal defect with pulmonary hypertension	Moderate; 12 months	Moderate; 6 months	80/26	Closure; atrial defect	Hypotension and acidosis (pH 7.15)	2	4	Recovery
Case 2	G. L. 14, M	Ventricular septal defect with pulmonary hypertension	Minimal	No	90 systolic	Closure; ventricular defect	Hypotension and dyspnea	2	1	Recovery
Case 3	V. C. 21, F	Tetralogy of Fallot	Moderate	No	—	Correction of tetralogy of Fallot	Dyspnea, anoxemia, low cardiac output	2	6	Recovery
Case 4	B. B. 19, F	Tetralogy of Fallot	Marked	No	—	Correction of tetralogy of Fallot	Dyspnea, hypotension and acidosis	1	11	Died from pneumonia
Case 5	J. F. 37, M	Mitral insufficiency	Severe	Severe	53/36	Repair; mitral insufficiency	Severe dyspnea	7	3	Recovery
Case 6	A. R. 54, F	Mitral stenosis	Severe	Severe	61/28	Mitral valvulotomy	Severe dyspnea	7	5	Recovery
Case 7	V. G. 43, F	Mitral stenosis, postoperative hemiplegia	Severe	Severe	50 (mean)	Mitral valvulotomy	Dyspnea, hypotension, and coma	1	43	Recovery
Case 8	B. L. 56, M	"Flail" chest from multiple rib fractures	Severe	No	—	—	Dyspnea, acidosis	1	21	Recovery
Case 9	A. H. 54, M	Herniation of heart following pneumonectomy	No	No	—	Radical right pneumonectomy	Hypotension and dyspnea with cardiac herniation into right pleural cavity	2	4	Recovery
Case 10	C. W. 44, F	Fibrothorax with pulmonary insufficiency, decortication	Severe	Mild	90/27	Decortication, left lung	Dyspnea, coma, and hypotension	3	41	Recovery

diffuse tracheobronchitis, however, and the possibility cannot be excluded that a pneumonia was the initial factor requiring aid from the respirator.

A possible disadvantage of a positive pressure respirator is the harmful effect on cardiac output. Studies by Cathcart and associates,[4] confirmed by work of Maloney,[5] and of Cournand and associates[6] have shown a progressive decrease in cardiac output as the mean endotracheal pressure is increased. We did not recognize any significant changes in cardiac output in the patients reported here when the respirator was used as described. Cardiac output was measured in 2 patients (Cases 3 and 4, Table I) and was not found to change significantly when the respirator was started. In 3 other patients not included in this series, no significant changes in cardiac output occurred with the use of the respirator. The necessity for a tracheotomy when the respirator is used is not a significant disadvantage. Most patients with a degree of pulmonary insufficiency sufficient to require a respirator are unable to remove bronchial secretions properly; so a tracheotomy is required for this reason alone.

CASE REPORTS

The 10 cases in which the respirator was used are listed in Table I. The 7 patients with cardiac disease included 1 patient with an atrial septal defect and severe pulmonary hypertension, 1 with a ventricular septal defect and moderate pulmonary hypertension, 2 patients with tetralogy of Fallot, and 3 with disease of the mitral valve, all 3 of whom had moderate pulmonary hypertension. The other 3 cases included 1 with a flail chest from multiple rib fractures, 1 with repeated herniation of the heart into the right pleural cavity after a radical pneumonectomy, and 1 with respiratory insufficiency from a fibrothorax of 13 years' duration. The respirator was used for intervals from 1 to 42 days. All but one recovered.

CASE 1.—E. P., a 34-year-old woman, had dyspnea and ankle edema for 1 year. Cardiac catheterization showed an atrial septal defect with a pulmonary artery pressure of 80/28 mm. Hg. The pulmonary blood flow was 1.6 times the systemic, and pulmonary arterial blood was 79 per cent saturated; pulmonary vascular resistance was 0.38 of the systemic resistance. The atrial defect was sutured during cardiopulmonary bypass on Feb. 1, 1958, through a right intercostal incision. Her course is charted in Fig. 2. Although her immediate postoperative condition was satisfactory (systolic blood pressure 100 mm. Hg, arterial blood pH 7.44, blood volume 80 ml. per kilogram), a progressive hypotension and vasoconstriction developed in the next 18 hours with a severe metabolic acidosis. Twelve grams of sodium bicarbonate were given intravenously for blood pH values as low as 7.15. An infusion of norepinephrine was used to maintain the systolic blood pressure at 90 mm. Hg. The patient became comatose and increasingly dyspneic, although there was no cyanosis and the chest roentgenogram showed no gross abnormality. She appeared in extremis 20 hours after operation. As a last resort, tracheotomy was done and the respirator connected when the arterial pH was 7.22. Her condition promptly improved with the respirator, the pH rising to 7.28 and then to 7.60 12 hours later. The vasopressor drug was gradually stopped over the next 18 hours as the patient's condition steadily improved. The respirator was gradually stopped over the fourth and fifth postoperative days and subsequent convalescence was uneventful.

Comment: Severe hypotension and metabolic acidosis subsided with the use of a respirator in a patient with increased pulmonary resistance and an atrial septal defect.

CASE 2.—G. L., aged 14, had repeated respiratory infections and growth retardation from a ventricular septal defect but had no dyspnea or congestive failure. The pulmonary artery systolic pressure was 90 mm. Hg and the pulmonary blood flow 11 L. per minute. On Aug. 21, 1957, the ventricular septal defect was sutured during cardiopulmonary bypass through a bilateral anterior thoracotomy. The pulmonary artery systolic pressure fell with closure of the defect from 80 to 53 mm. Hg. Twelve hours after operation, an acute hypotension began that soon was associated with a serum sodium of 125 mEq. per liter and hemoconcentration with an hematocrit of 54. The hypotension was refractory to vasopressor drugs but responded to hypertonic saline. Progressive dyspnea on the second postoperative day was not relieved by a tracheotomy, so the Mörch respirator was begun 4 hours later. Dyspnea was promptly relieved, and the hypotension improved. The pH

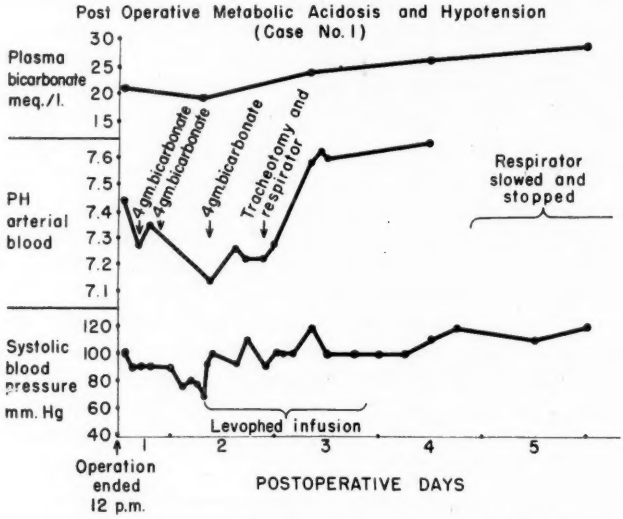

Fig. 2.—The response of postoperative hypotension and metabolic acidosis in Case 1 to a respirator is shown.

3 hours later was 7.58. He improved rapidly and within 12 hours the respirator and vasopressor were stopped. He remained in moderate congestive failure with elevated venous pressure and hepatomegaly for the next several days but gradually improved and was discharged in good condition on the twentieth postoperative day.

Comment: Hypotension and dyspnea, probably from cardiac failure, responded promptly to the use of the respirator after repair of a ventricular septal defect.

CASE 3.—V. C., aged 21, had moderate dyspnea from a tetralogy of Fallot with cyanosis and an hematocrit of 50 per cent. The ventricular septal defect was closed and an infundibular stenosis excised on Oct. 15, 1958, with cardiopulmonary bypass and a median sternotomy. The right ventricular systolic pressure fell from 90 to 30 mm. Hg. Her postoperative course is charted in Fig. 3. Progessive dyspnea and hypotension gradually developed over the next 8 hours. Cardiac output was estimated at intervals from oxygen consumption and blood samples obtained through small plastic catheters in the pulmonary and femoral arteries; this showed a striking reduction of cardiac index, 1.5 L. per minute 8 hours after operation, with an arterial oxygen saturation of 94 per cent. The arterial oxygen saturation subsequently decreased to 82 per cent in spite of an oxygen tent. A tracheotomy was done 12 hours after operation with a rise in oxygen saturation to 88 per cent; dyspnea remained, however, and

the Mörch respirator was started at this time. The arterial oxygen saturation rose to 93 per cent and her condition steadily improved. Several hours later the cardiac index had risen to 2.8 L. per minute and the arterial oxygen saturation was 96 per cent. Throughout this period the pH of the arterial blood ranged from 7.36 to 7.58. The respirator was gradually stopped on the sixth and seventh postoperative days and her subsequent convalescence was uneventful.

Comment: The cause of the low cardiac output, anoxemia, and ventilatory insufficiency is unknown in this patient with a tetralogy of Fallot. The response to the respirator was prompt and sustained.

CASE 4.—B. B., a 19-year-old girl, had marked limitation from a tetralogy of Fallot with dyspnea, cyanosis, and an hematocrit of 74 per cent. A left subclavian-pulmonary artery anastomosis had been performed in 1945. On Nov. 7, 1958, the ventricular septal defect was closed, the infundibular stenosis excised, and the subclavian-pulmonary anastomosis closed through a median sternotomy and with cardiopulmonary bypass. The right ventricular systolic pressure decreased from 145 to 32 mm. Hg. Six hours after operation the blood

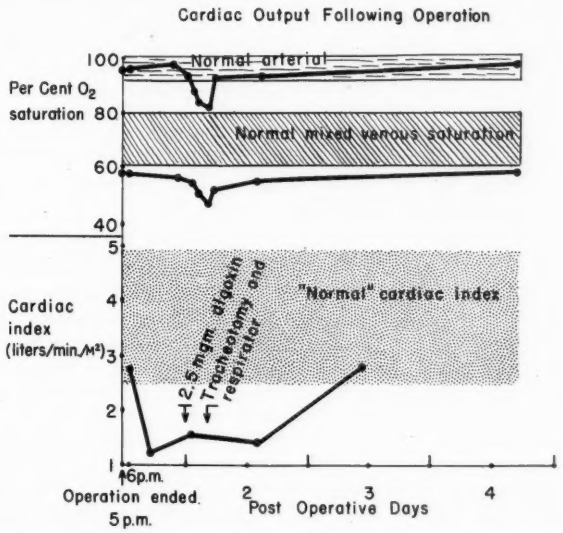

Fig. 3.—The response of postoperative anoxemia and low cardiac output in Case 3 to a respirator is shown.

pressure was 70/50 mm. Hg; she was dyspneic and slightly acidotic (pH 7.31). A tracheotomy was done and the respirator begun. Dyspnea was satisfactorily controlled by the respirator, but throughout the remainder of her hospital course she was unable to ventilate satisfactorily without it. Samples of mixed venous blood obtained through a small plastic catheter left in the pulmonary artery at the time of thoracotomy showed a low oxygen content, indicating a low cardiac output. This persisted and never reached a normal level. After the seventh postoperative day, resistance to ventilation progressively increased; bronchial secretions were profuse, purulent, and removed with difficulty. Several episodes of acute ventilatory obstruction were relieved by vigorous suctioning and by bronchoscopy on one occasion. The episodes of respiratory obstruction progressively increased in severity and the patient died on the eleventh postoperative day. Autopsy showed diffuse purulent tracheobronchitis with pneumonia involving all lobes of both lungs.

Comment: Dyspnea, hypotension, and metabolic acidosis were present soon after correction of the tetralogy of Fallot. Respiratory insufficiency required

the constant use of the respirator for 11 days before the patient died of a diffuse tracheobronchitis.

CASE 5.—J. F., aged 37, had mitral insufficiency with severe congestive heart failure, hepatomegaly, and pedal edema. Cardiac catheterization showed a pulmonary artery pressure of 53/36 mm. Hg and a left atrial pressure of 50/20 mm. Hg. The mitral valve was repaired with cardiopulmonary bypass through a right thoracotomy on Dec. 22, 1958. The insufficiency came from two perforations in the aortic leaflet of the mitral valve, probably a result of bacterial endocarditis. After the perforations were sutured, the mitral valve seemed competent; subsequent left atrial pressures were 10 to 15 mm. Hg. Re-operation was necessary on the third postoperative day because of persistent bleeding from a lacerated internal mammary vein. Following the second thoracotomy, dyspnea progressively increased in severity. Bronchial secretions were profuse with numerous râles throughout the chest. The patient became disoriented, with a blood urea nitrogen above 40 mg. per cent. A tracheotomy on the seventh postoperative day resulted in some improvement; the arterial blood pH after tracheotomy was 7.30. Severe dyspnea remained, however, and several hours later the respirator was begun. This promptly controlled the dyspnea and his condition became stable. The serum bicarbonate was 32 mEq. per liter 8 hours after the respirator was started and fell to 27 mEq. per liter 24 hours later. The respirator was used for 3 days, after which it was gradually stopped over a 24-hour period. The patient was discharged in good condition on the twenty-eighth postoperative day.

Comment: Cardiac failure, profuse bronchial secretions, and fatigue resulted in marked dyspnea, respiratory acidosis, and azotemia which were controlled by the use of the respirator for 3 days.

CASE 6.—A. R., a 54-year-old woman, had mitral stenosis and insufficiency with dyspnea, hepatomegaly, and ankle edema. She had spontaneously recovered from a massive arterial embolus to the lower extremities 1 year earlier, but femoral pulses were not palpable and bilateral intermittent claudication was present. Cardiac catheterization showed a pulmonary artery pressure of 61/28 mm. Hg and a left atrial pressure of 26/17 mm. Hg; the end diastolic gradient across the mitral valves was 5 mm. Hg. Because mitral insufficiency was considered the dominant defect and also because of the previous arterial embolism she was operated upon through a right posterolateral incision with cardiopulmonary bypass on May 2, 1958. A stenosed mitral valve, less than 1 cm. in diameter, was found in which there was little insufficiency. Both commissures were split to produce an opening over 2 cm. in diameter with little change in the regurgitation. A thrombus was removed from the left atrial appendage and the appendage closed from within the left atrium. The left atrial systolic pressure remained in the range of 25 to 30 mm. Hg after operation. Cardiac failure became progressively more severe in the first few days after operation. On the second postoperative day, the arterial blood pH was 7.36 and the plasma bicarbonate 26 mEq. per liter (Table II). On the fourth postoperative day dyspnea was more severe, with episodes of mental

TABLE II. CASE 6. BLOOD CHEMICAL CHANGES

POSTOPERATIVE DAY	ARTERIAL pH	SERUM BICARBONATE (mEq./L.)	BLOOD UREA NITROGEN (mg. %)
1	7.41; 7.34	27	30
2	7.36	26	32
4		28	50
7		31	64
Respirator begun			
4 hours later	7.58		
10 hours later	7.60		
8	7.7; 7.57	33	
9		31	26

confusion; plasma bicarbonate was 28 mEq. per liter. On the seventh postoperative day dyspnea was so extreme that the patient was comatose with ineffective gasping respiration; a plasma bicarbonate was 31 mEq. per liter and blood urea nitrogen was 64 mg. per cent. A tracheotomy was done and the Mörch respirator begun. Ten hours later, the pH of arterial blood was 7.6; the following day the pH was 7.7 and subsequently 7.57; plasma bicarbonate was 33 mEq. per liter. Forty-eight hours after the respirator was begun the patient was alert and responsive; the urea nitrogen had fallen to 26 mg. per cent. The respirator was gradually stopped between the ninth and the eleventh postoperative days and the patient was discharged in good condition on the twenty-eighth postoperative day.

Comment: Cardiac and respiratory failure, resulting in coma, respiratory acidosis, and azotemia, were controlled with the use of the respirator for 4 days. An earlier use of the respirator was probably indicated.

CASE 7.—V. G., a 43-year-old woman, had progressive cardiac failure from mitral valve disease with dyspnea, hepatomegaly, and ankle edema. Mitral insufficiency was thought to be the predominant lesion. On Feb. 28, 1958, through a right anterolateral incision, cardiopulmonary bypass was used to allow visual examination of the mitral valve. The initial diagnosis proved to be incorrect, however, as severe mitral stenosis was found with a valve opening only 5 to 6 mm. in diameter. Tricuspid insufficiency was present but no mitral insufficiency. A satisfactory commissurotomy was performed which produced minimal regurgitation. A large thrombus was removed from one of the pulmonary veins during the procedure.

A left hemiplegia was noted on recovery from anesthesia, and ventilation was inadequate. A tracheotomy was done and the Mörch respirator applied. The first 3 days after operation were characterized by coma, hypotension, elevated venous pressure, and oliguria; vasopressor drugs were used for 48 hours. The arterial blood pH ranged from 7.43 to 7.57 in the first 4 days, and the arterial oxygen saturation 3 days after operation was 99 per cent.

The patient remained comatose with severe cardiac failure for several weeks. The liver was enlarged below the umbilicus and peripheral edema was present. Attempts to stop the respirator resulted in marked cyanosis and dyspnea until 38 days after operation. She was subsequently taken off the respirator for an increasingly longer period until it was stopped completely on the forty-third day. Gradual improvement of the hemiplegia occurred and when she was discharged from the hospital 2 months after operation she was able to walk and had partial use of her left arm.

Comment: Respiratory insufficiency resulting from cardiac failure and hemiplegia would surely have been fatal without the prolonged use of the respirator.

CASE 8.—B. L., age 56, was hospitalized on Dec. 11, 1956, following a crushing injury to the chest in an automobile accident. All 12 ribs in the left hemithorax were fractured, and the first, eighth, and ninth ribs in the right (Fig. 4). Moderate subcutaneous emphysema was present but no pneumothorax. Only a moderate instability of the chest wall was present, but the patient gradually became more and more dyspneic. His subsequent course is charted in Fig. 5. A tracheotomy 12 hours after admission did not relieve the dyspnea. Analysis of arterial blood showed a pH of 7.15, an oxygen saturation of 77 per cent, and a carbon dioxide content of 51 volumes per cent. Artificial respiration was begun with the Mörch respirator with rapid improvement in the patient's color and general condition. Twenty-four hours later the arterial blood pH was 7.55, the carbon dioxide content 47 volumes per cent and the oxygen saturation 94 per cent. Subsequent determinations were within normal limits. The respirator was gradually stopped from the fourteenth to the twenty-first day following the injury, and the patient was discharged in good condition 30 days following injury (Fig. 6).

Comment: Severe respiratory insufficiency from a flail chest was not relieved by tracheotomy but was promptly controlled with the respirator.

CASE 9.—A. H., age 54, had a radical right pneumonectomy on Sept. 3, 1958, for a large bronchogenic carcinoma. A large amount of pericardium was removed and the pericardial sac left in communication with the right pleural cavity. Two additional thoracotomies were required in the next 24 hours for shock resulting from herniation of the heart into the right pleural cavity. This herniation was precipitated by periods of dyspnea and restlessness during which time the patient would not remain on his left side as

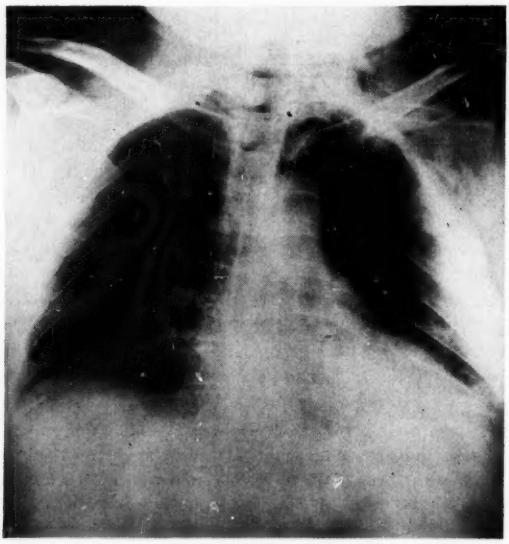

Fig. 4.—Chest roentgenogram following trauma in Case 8. All twelve ribs in the left chest are fractured, as well as ribs 1, 8, and 9 on the right.

prescribed. After the herniated heart had been replaced the third time, a tracheotomy was performed and the Mörch respirator begun. The respirator controlled the dyspnea and restlessness, and it was possible to keep him comfortably on his left side for 4 days, after which the respirator was stopped and further respiratory insufficiency did not occur.

Comment: Dyspnea and hypotension in this patient prevented proper positioning to avoid herniation of the heart until respirations were controlled with a respirator.

CASE 10.—C. W., age 44, had severe respiratory insufficiency from a fibrothorax which followed pneumothorax therapy for tuberculosis 13 years before. She was totally incapacitated with dyspnea and moderate right heart failure. The vital capacity was 1,400 ml., the maximum breathing capacity 51 L. per minute, the arterial oxygen saturation 80 per cent, and the pulmonary artery systolic pressure 90 mm. Hg. After many preoperative studies and much deliberation, a decortication was undertaken on Mar. 15, 1957. A complete decortication was possible, but a massive hemothorax caused increasing respiratory insufficiency over the next 48 hours. At this time the patient almost died in acute respiratory failure, but she was resuscitated with an emergency tracheotomy, evacuation of the hemothorax, and maintenance of the respirations with a respirator. After the respirator had been

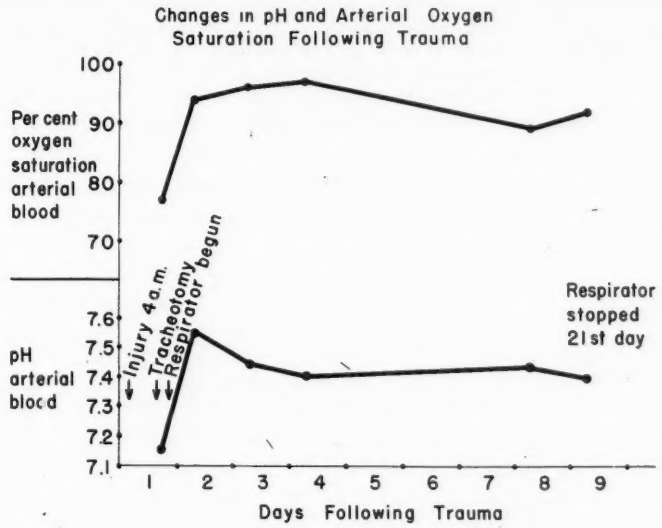

Fig. 5.—The response to a respirator of anoxemia and respiratory acidosis caused by a flail chest in Case 8 is shown. The response to tracheotomy without a respirator was not satisfactory.

Fig 6.—Chest roentgenogram 18 days following injury in Case 8. The subcutaneous emphysema has subsided; the respirator was stopped on the twenty-first day.

used for 48 hours the arterial oxygen saturation was 85 per cent, the pCO_2 40 mm. Hg, and the pH 7.28. Attempts to stop the respirator over subsequent days resulted in severe dyspnea and cyanosis with arterial oxygen saturations of 30 to 45 per cent. The respirator was constantly required for 5 weeks, after which it was gradually stopped by the forty-first postoperative day. When discharged from the hospital 2½ months after operation, oxygen saturation was only 75 per cent, pCO_2 35 mm. Hg, and the pH 7.42. The patient's exercise tolerance steadily improved, however, and 6 months later she was able to walk 18 blocks with no dyspnea whatever.

Comment: Because of severe respiratory insufficiency, a respirator was needed for this patient for 6 weeks following decortication of a long-standing fibrothorax.

DISCUSSION

Etiology of Respiratory Insufficiency.—The respiratory insufficiency in the 3 patients with pulmonary disease (Cases 8, 9, and 10, Table I), was caused by the mechanical effects of the disease present—a flail chest, a pneumonectomy and cardiac herniation, and a chronic fibrothorax, respectively. The cause of the insufficiency is less obvious, however, in the 7 patients with cardiac disease. Cardiac failure, pulmonary hypertension, and temporary decrease in pulmonary function from thoracotomy are possible causative factors. Patients with chronic cardiac failure, which was present in 3 patients with mitral valve disease (Cases 5, 6, and 7, Table I), often have severe cardiac failure after mitral commissurotomy. This tendency is well shown by the stormy, and often fatal, postoperative course following digital commissurotomy for mitral stenosis in patients with intractable congestive failure (so-called Class 4 patients).[7]

Pulmonary hypertension, which was present in 5 of the 7 patients, is also often associated with postoperative difficulties with ventilation and hypotension. These difficulties may arise from the fact that anoxia markedly increases pulmonary vascular resistance. A degree of anoxia easily tolerated by a patient whose pulmonary artery pressure is normal may be disastrous to the patient with severe pulmonary hypertension.

The cause of the ventilatory insufficiency in the 2 patients with tetralogy of Fallot (Cases 3 and 4, Table I) is not known. Neither had cardiac failure or pulmonary hypertension before operation. Both had a marked decrease in cardiac output after operation from unknown causes: 1, No. 3, had potassium asystole during bypass; the other did not. Experimental studies of pulmonary function following cardiopulmonary bypass by Schramel, Creech, and associates[8] have shown a decreased pulmonary diffusion capacity for several days after operation. The cause at present is unknown.

Indications for Use of Respirator.—Dyspnea, progressing to physical exhaustion and coma in some patients, was the clinical indication for the use of a respirator in the 3 patients with mitral valve disease (Cases 5, 6, and 7) and the 3 patients with pulmonary disease (Cases 8, 9, and 10). Severe hypotension and metabolic acidosis, not responding to blood transfusions and vasopressors,

were the indications for the respirator in 2 patients (Cases 1 and 2). Moderate dyspnea was also present. A respirator was used for the 2 patients with tetralogy of Fallot (Cases 3 and 4) because of a combination of dyspnea, arterial oxygen unsaturation, and low cardiac output.

The respirator was used only after other simpler measures had failed. These measures included high humidity, endotracheal suction to remove secretions, bronchodilators, sometimes aminophylline and hydrocortisone. Cardiac failure was treated with digitalis, diuretics, and fluid restriction, and measurement and regulation of blood volume. Attempts to manage the patients with tracheotomy alone were used in most instances, and the respirator begun only when this was unsatisfactory. Our hesitation to use the respirator is evident from the fact that some of the patients were drowsy or comatose, hypotensive and sweating, with ineffectual respiration, when the respirator was started. Although improvement may be dramatic under such circumstances, the respirator probably should have been used earlier.

Results.—Nine of the 10 patients made a satisfactory recovery. The respiratory insufficiency was so severe in all of these patients that survival without the use of the respirator was considered unlikely. The origin of the fatal pneumonia in Case 4 is unknown. Aseptic precautions were routinely carefully observed in the management of patients on the respirator. These included washing of the hands before tracheal aspiration and the use of sterile catheters. Serious respiratory infection did not develop in any of the 61 patients treated with a respirator by Björk and Engström.[3]

The favorable outcome in 6 of the 7 patients with cardiac disease in this series was due to the fact that the respiratory insufficiency was due to reversible causes. We have unsuccessfully used the respirator in a few other patients following cardiopulmonary bypass who were seriously ill with hypotension and coma in whom respiratory insufficiency was not a prominent feature. Although respiratory function was sustained in these patients, circulatory failure and coma persisted and ended fatally.

Our present concept of the value of the respirator is to support the patient during a period of transient decrease in respiratory function. If the respiratory insufficiency is secondary to circulatory failure that does not improve, the respirator will not be of value. Patients who may most frequently benefit from the respirator following operation are patients with pulmonary hypertension, often found with ventricular septal defect, and patients with mitral or aortic valvular disease with chronic cardiac failure before operation.

SUMMARY

Ten patients with severe respiratory insufficiency were treated with a Mörch piston respirator from 1 to 42 days. Nine of the 10 patients recovered.

The respiratory insufficiency resulted from chest wall trauma in 1 patient, followed pulmonary surgery in 2 patients, and followed cardiac surgery with

cardiopulmonary bypass in 7 patients. In these 7 patients the cause of respiratory insufficiency was often uncertain. Chronic congestive failure and pulmonary hypertension before operation were the most likely causes in some of the group, but in others the cause was unknown.

A respirator has not been frequently used for respiratory insufficiency resulting from cardiac disease. The experiences presented here suggest that postoperative respiratory insufficiency following cardiac surgery may be successfully treated in some patients with a respirator until recovery of respiratory function occurs.

REFERENCES

1. Björk, V. O., and Engström, C. G.: The Treatment of Ventilatory Insufficiency After Pulmonary Resection With Tracheostomy and Prolonged Artificial Ventilation, J. THORACIC SURG. **30**: 356, 1955.
2. Avery, E. E., Mörch, E. T., and Benson, D. W.: Critically Crushed Chests, J. THORACIC SURG. **32**: 291, 1956.
3. Björk, V. O., and Engström, C. G.: The Treatment of Ventilatory Insufficiency by Tracheostomy and Artificial Ventilation, J. THORACIC SURG. **34**: 228, 1957.
4. Cathcart, R. T., Nealon, T. F., Fraimow, W., Hampton, L. J., and Gibbon, J. H.: Cardiac Output Under General Anesthesia, Ann. Surg. **148**: 488, 1958.
5. Maloney, J. V., Elam, J. O., Handford, S. W., Balla, S. W., Eastwood, D. W., Brown, E. S., and Ten Pas, R. H.: Importance of Negative Pressure Phase in Mechanical Respiration J. A. M. A. **152**: 212, 1953.
6. Cournand, A., Motley, H. L., Werko, L., and Dickinson, W. R.: Physiological Studies of the Effects of Intermittent Positive Pressure Breathing on Cardiac Output in Man, Am. J. Physiol. **152**: 162, 1948.
7. Ellis, L. B., Abelmann, W. H., and Harken, D. E.: Selection of Patients for Mitral and Aortic Valvuloplasty, Circulation **15**: 924, 1957.
8. Schramel, R. J., Cameron, R., Ziskind, M., Adam, M., and Creech, O.: Studies of Pulmonary Diffusion After Open Heart Surgery, J. THORACIC SURG. **38**: 281, 1959.

(There was no discussion of this paper.)

THE INTRATHORACIC COMPLICATIONS OF SUBPHRENIC ABSCESS

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OVER 100 patients with subdiaphragmatic space infection have been treated at the Lahey Clinic during the past 10 years. An interesting group of problems has resulted, due to a large surgical service, which included complex and secondary operations on the biliary tract, pancreas, stomach, and spleen. A previous article[2] dealt with some changing concepts relative to the anatomy and pathology of the subphrenic spaces. These concepts will be briefly reviewed before the complications of this disease are considered here.

THE ANATOMY OF THE SUBPHRENIC SPACES

The time-honored anatomic and pathologic concepts of Barnard[1] have been widely accepted[3, 4] and are to be found in standard textbooks of surgery. From the anatomic standpoint it is commonly believed that the coronary ligament divides the superior surface of the liver into anterior and posterior compartments. Thus, anterior and posterior spaces are described on both sides and the nomenclature becomes cumbersome and confusing. The basis for this confusion is the fact that spaces are described which do not exist.

Fig. 1 shows how this misconception regarding the position of the coronary ligament causes confusion. The important consideration is that the coronary ligament suspends the liver not from above, as shown in this drawing, but from the dorsal aspect of the abdominal wall.

Fig. 2 shows the anatomic relationships of the suprahepatic area as we understand them. Thus, on the right side there are only two spaces—one above the liver and one below. The space which is usually designated "right posterior superior" does not exist. This can be readily confirmed at laparotomy or in the dissecting room. Since the right posterior superior subphrenic space is considered the most common site of infection, the importance of this point of confusion is apparent.

Fig. 3, a cross-sectional drawing, shows how the misapprehension may be further propagated. In this drawing, the coronary ligament and the bare area are shown in a superior position, thus giving rise to the appearance of an anterior and posterior space on the right and left sides. Study in the anatomy laboratory shows that no such space exists on either side.

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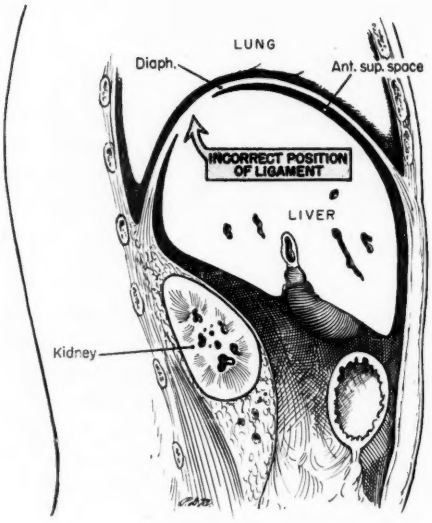

Fig. 1.—*For legend see opposite page.*

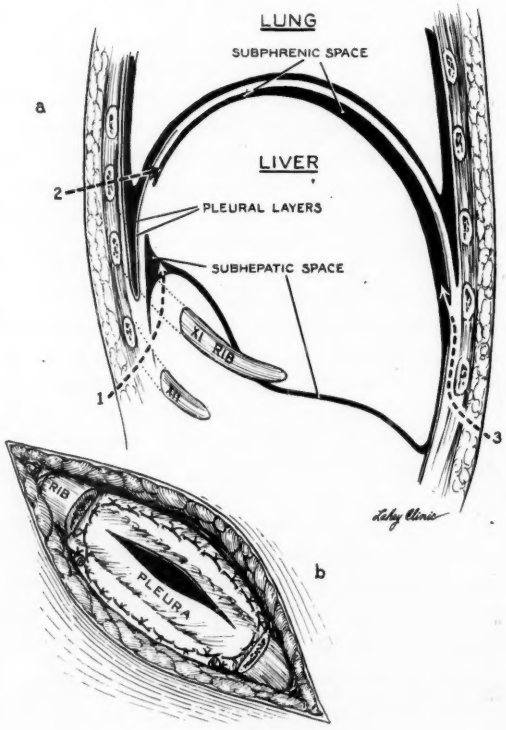

Fig. 2.—*For legend see opposite page.*

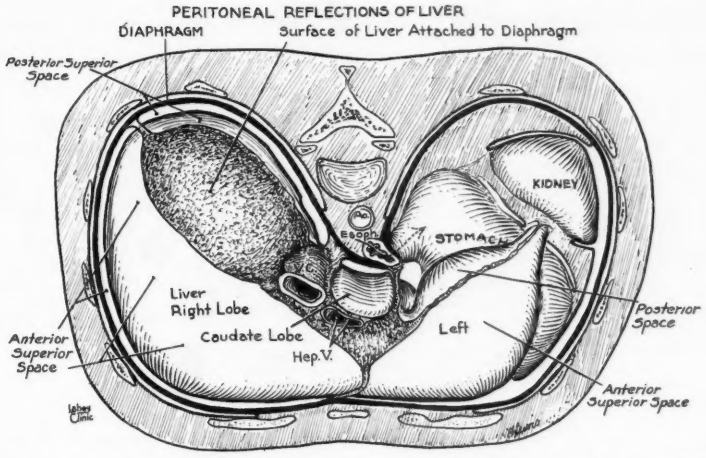

Fig. 3.—Usual type of drawing suggesting the presence anatomically of anterior and posterior spaces on both sides. The spaces indicated above and designated "posterior superior space" on the right and "posterior space" on the left do not exist. Actually, the bare area and both triangular ligaments are considerably more posterior than the diagram suggests.

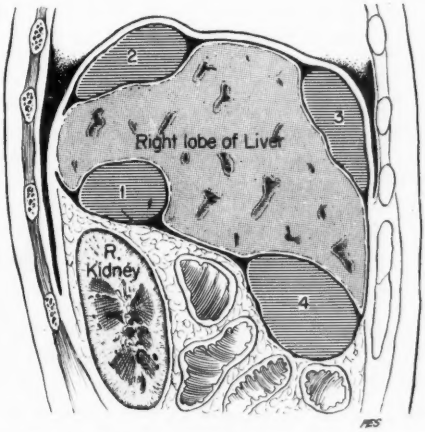

Fig. 4.—*1*, Right posterior subhepatic abscess. This site may be readily drained extrapleurally and extraperitoneally below or through the bed of the resected twelfth rib (Ochsner: Fig. 2. *1*). This type of collection is often termed "right posterior subphrenic" in standard texts.

2, Right posterior subphrenic abscess. It is obvious now, when the anatomic relations of the coronary ligament are carefully drawn, that this abscess is considerably above the pleural reflection. Therefore, it can be drained only by some modification of the Trendelenburg approach.

3, Right anterior subphrenic abscess. The approach to this part of the subdiaphragmatic space is a subcostal incision on the right going extraperitoneally and extrapleurally (Ochsner).

4, Typical right anterior subhepatic abscess easily reached through the existing right rectus or subcostal incision.

Fig. 1.—The right triangular ligament is usually depicted as dividing the superior surface of the liver into anterior and posterior spaces. This error is sometimes even more exaggerated than shown above. The proper anatomic relationships are shown in Fig. 2.

Fig. 2.—The very large areas occupied by the subphrenic and subhepatic spaces on the right side are shown. *a*, The various approaches to these spaces are indicated: *1*, twelfth rib extraserous route (Ochsner); *2*, Trendelenburg transpleural route. The technique of suturing the pleura to the diaphragm is shown in insert *b*.

PATHOLOGIC ANATOMY

From the clinical and pathologic standpoint, the above remarks regarding anatomy represent an oversimplification. The normal anatomy should be clearly understood before pathology is considered, however. The presence of loculations in the subphrenic and subhepatic areas makes it essential that the site of infection be localized more precisely.

It has been emphasized that the subphrenic and subhepatic spaces are large, particularly on the right side. Characteristically, these large spaces become subdivided about their mid-points by the formation of pyogenic membranes. We are therefore compelled to acknowledge the presence of anterior and posterior subphrenic and anterior and posterior subhepatic abscesses. Fig. 4 shows typical collections.

COMPLICATIONS

The complications of subphrenic space infection are chiefly intrathoracic, since the septic process frequently spreads through the diaphragm into the pleural cavity or into the lung. Purulent exudate has a tendency to try to escape, and the soft diaphragm and lung constitute a less formidable barrier than do the rigid liver and posterior parietal structures. In 1 case, however, purulent exudate from an anterior subphrenic abscess on the right side perforated into the general abdominal cavity with diffuse peritonitis and rapid fatality.

The intrathoracic complications of subdiaphragmatic abscess will be discussed briefly under three headings: (1) serous effusion, (2) purulent subphrenic infection with perforation into the thorax, and (3) obstruction of the biliary tract with subdiaphragmatic biliary abscess and intrathoracic communication.

Serous Effusion.—

We have been interested in the nature and characteristics of the serous effusion which so frequently accompanies subdiaphragmatic abscess. We have learned that it is usually ipsilateral, but not always. It is seldom massive in amount unless cardiac disease is also present. It may be in evidence with subhepatic infection, but this is unusual. In most instances it indicates infection above the liver.

In a few instances, we have had the opportunity to study this fluid and have found that it is clear, pale yellow, and has a high neutrophil and protein content. It has always been sterile on culture. We were not able to determine any particular point of onset of this fluid in the course of subdiaphragmatic infection. It has not proved to be a therapeutic problem in any way.

Subphrenic Infection With Intrathoracic Communication.—

If the pleural cavity is free, perforation of purulent exudate into the chest produces empyema. This may be massive and result in very rapid toxicity; thus, unless prompt drainage of both thorax and subphrenic area is provided,

death may ensue. In our experience, when prompt drainage of both spaces has been carried out, the lung has rapidly re-expanded and healing has occurred.

In the cases in which pleural symphysis is present, however, a fistula forms between the subphrenic area and the bronchial tree. This condition may follow one of two general forms. An acute fulminating type of necrotizing bronchopneumonia may occur, with rapid toxicity and death, or the course may be extremely indolent. The following case reports illustrate these categories.

A 42-year-old man was admitted to the hospital in June, 1944, with the diagnosis of carcinoma of the pancreas. This diagnosis was not substantiated at laparotomy, and the abdomen was closed. Convalescence was smooth and the patient was discharged 2 weeks

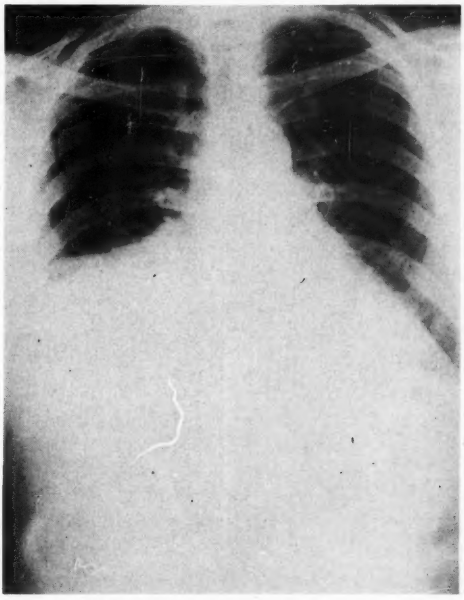

Fig. 5.—Marked elevation of right leaf of diaphragm in a patient with perforation of a subphrenic abscess into the bronchial system.

following operation. Two years later, in July, 1956, he was readmitted. He had been in the hospital at his home town for a week or two with unexplained fever and, on the day before admission to the Lahey Clinic, had experienced a sudden episode of copious expectoration with rapid development of dyspnea and toxicity.

The clinical picture and the x-ray studies (Fig. 5) suggested subdiaphragmatic abscess on the right side with perforation into the right lung and bronchial tree. Drainage of the subphrenic space was carried out, followed by tracheostomy. The patient improved for a few hours but became increasingly ill and died.

A number of these patients have survived, but this case is cited because of the fact that a postmortem examination was obtained. This revealed chronic pancreatitis without any evidence of purulent exudate anywhere except in the right suprahepatic space. This space had been adequately drained.

This case also illustrates the chronicity which is possible in subphrenic abscess. A purulent collection had presumably been present above the right lobe of the liver for 2 years without symptoms before perforation into the thorax occurred. It also demonstrates the severity of the resulting necrotizing bronchitis and bronchopneumonia.

Perforation into the chest may not be such a dramatic phenomenon in some cases, however, and may be almost of the occult variety.

An 18-year-old boy was seen at the clinic following perforation of an ileostomy with a catheter. The stoma was revised, and an attempt made to rinse out the abdominal cavity as well as possible.

A few months later, this boy was admitted with fever and leukocytosis. He stated that he had failed to gain weight. Roentgenographic examination was strongly suggestive of right subphrenic abscess (Fig. 6). This abscess was found and drained. During convalescence, Lipiodol was instilled into the sinus above the right lobe of the liver in order to determine its size, and filling of the bronchial tree resulted (Fig. 7).

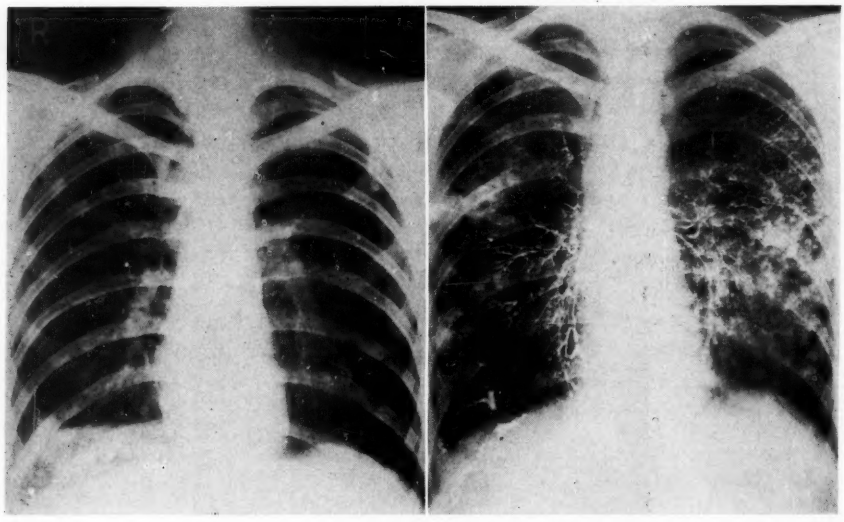

Fig. 6. Fig. 7.

Fig. 6.—High fixed diaphragm with air fluid levels on right, indicating subphrenic abscess.
Fig. 7.—Instillation of dye into subphrenic space with filling of bronchial tree, indicating perforation of a subphrenic abscess into the lung.

Experience has shown that when drainage of the subphrenic area is carried out, the pulmonary lesion will usually heal. In most cases, lobectomy will not be necessary.

Biliary Communications With the Intrathoracic Cavity.—

In the course of our work with postoperative strictures of the bile ducts, we have encountered a number of patients in whom, following secondary or complex operations, subdiaphragmatic collections of bile have developed. These collections in turn have, at times, perforated into the thorax. Again, two sets of circumstances are possible. If the pleura is free, a large collection of bile

accumulates in the intrathoracic cavity. This disturbs the intrathoracic dynamics and may cause tension unless evacuated promptly. Furthermore, secondary infection is almost sure to ensue. Management of these patients consists of correcting the intrathoracic dynamics by trocar drainage, followed by proper attention to the obstructing lesion in the biliary tract.

At times, however, pleural symphysis is present and when perforation occurs, it occurs into the bronchial tree, resulting in a bronchobiliary fistula. The type of pneumonitis and bronchitis which is found in this circumstance is a very serious one because of the necrotizing effect of bile. Prompt drainage of the subphrenic area is essential to stop this expectoration of bile. The lesion cannot be healed until the obstruction in the common duct has been removed, however.

GENERAL PRINCIPLES OF TREATMENT

Some of these principles have been implied in the paragraphs above. One special point relative to the site of drainage will be mentioned, however. The type of abscess which we see today is commonly a right posterior subphrenic one (Fig. 4, *2*). It will be seen from the drawing that this abscess is best reached by intrathoracic, transpleural drainage. The abscess designated *1* in Fig. 4 can, of course, be drained extraserously. This is not truly a subphrenic abscess at all, however; it is subhepatic. The type of abscess which we see following secondary operations on the upper abdominal viscera is different from the type of abscess which characteristically followed generalized peritonitis in former years.

SUMMARY AND CONCLUSIONS

In this brief presentation I have attempted to make three major points. (1) The anatomy of the diaphragm and the suprahepatic area can be made simple and clear. (2) Subphrenic abscess as we see it today is a more chronic and indolent affair than it has been in the past. Thus, it may be more dangerous from the standpoint of intrathoracic complications. (3) For cure of subdiaphragmatic abscess, as we now see it, a transpleural approach is required.

REFERENCES

1. Barnard, H. L.: An Address on Surgical Aspects of Subphrenic Abscess, Brit. M. J. 1: 371-429, 1908.
2. Boyd, D. P.: The Anatomy and Pathology of the Subphrenic Spaces, S. Clin. North America **38**: 619-626, 1958.
3. Faxon, H. H.: Subphrenic Abscess; Report of 111 Consecutive Operative Cases, New England J. Med. **222**: 289-299, 1940.
4. Ochsner, A., and Graves, A. M.: Subphrenic Abscess; Analysis of 3,372 Collected and Personal Cases, Ann. Surg. **98**: 961-990, 1933.

DISCUSSION

DR. LAURENCE H. RUBENSTEIN, Chicago, Ill.—I enjoyed the excellent presentation by Dr. Boyd, and I merely rise to confirm what he has said; namely, in order to cure the pulmonary complications one must first treat the subdiaphragmatic disease.

I have had experience with 3 patients who presented themselves with pulmonary problems. The outstanding symptom in each case was hemoptysis associated with mucopurulent sputum.

[Slide] The first case was a man who presented himself with suspected carcinoma of the lung with hemoptysis, mucopurulent sputum, weight loss, cachexia, and an opacity in the right lower lobe. An interesting finding on bronchoscopy was that the anterior medial basal segment was hyperemic and the pus seemed to be coming from that area.

In going back into his history, he had had previous biliary disease; jaundice the year before. At exploratory operation, he was found to have an absence of the gall bladder. On careful examination we found a slightly tender anterior bulge of the right lower chest. This turned out to be the subphrenic abscess, which was drained, and the patient made a complete recovery.

This roentgenogram is an anterior posteroanterior projection of the same patient showing the suspect right lower lobe involvement.

Another case was that of a woman who also presented herself with a primary picture of pulmonary disease, expectoration of pus, and blood. Again on bronchoscopy it was noted in this particular case that the anterior medial basal segment of the right lower lobe was also involved with hyperemia and pus coming from this area.

A history of gall bladder disease was elicited. A subphrenic abscess was suspected, discovered, and drained. The patient made a good recovery.

The next case—I am sorry I don't have a slide to show—was a most interesting case, because the patient was admitted as an emergency due to massive exsanguinating hemoptysis. An anterior biliary fistulous drainage tract in the epigastrium was closed. The etiology of the fistula was never thoroughly established. Subsequent to fistula closure, the patient coughed bile and developed massive hemoptysis which required a lingulectomy because the fistulous tract extended from the left lobe of the liver to the lingula. This was demonstrated by bronchogram preoperatively. A life-saving lingulectomy was done.

Subsequently, the patient has had an abdominal exploration. No true cause for the biliary fistula could be established. The origin of the fistula was from the anterior portion of the left lobe of the liver. A jejunobiliary anastomosis was made and the patient made a good recovery.

The pulmonary disease subsided only after the underlying subphrenic pathology was adequately treated.

DR. DUANE CARR, Memphis, Tenn.—I wish to thank Dr. Boyd for bringing a very important subject to our attention, and I would like particularly for the younger men to note how carefully Dr. Boyd sutured the pleura to the diaphragm *before* he crossed the pleural space. Of course it is impossible to suture it if one is going to put a trocar, needle, or what-not across the pleural space in searching for an abscess which he may be unfortunate enough to find by needle, thereby contaminating the pleural space very thoroughly. For that reason some of us who have dealt with these abscesses over a number of years hesitate to probe for them with needles.

What Dr. Boyd has said, with which I agree completely, applies to the pyogenic or pyrogenic infections, I think without exception. I would like to ask him what his attitude may be toward a surgical approach to the pure amebic subphrenic infections, including liver abscesses. In our experience we have not done nearly so well with these by a surgical approach as we have with the pyogenic organisms.

DR. ADRIAN LAMBERT, New York, N. Y.—I recently had occasion to summarize the cases of subphrenic abscesses at Roosevelt Hospital between 1940 and 1957. In this group of 29 patients on whom 33 procedures were done the relevant fact is that 93 per cent of these patients presented themselves with pulmonary complications, including an elevated diaphragm in 78 per cent, atelectasis in 63 per cent, pain and pleurisy in 56 per cent, and the presence of chest fluid in 50 per cent.

The second point that I think is of interest is that in these patients the interval of time between the development of the subphrenic abscess and the time that they were drained was twice as long in those who had antibiotics as in those who didn't, showing that the antibiotics will mask the symptoms of subphrenic abscess; and the usual pain, splinting of the abdomen and so forth are not present when you have the patient under antibiotic therapy.

Two empyemas developed in this group, and we felt that they developed secondary to traumatic thoracentesis where the needle had been introduced into the abdomen through the pleural space, so that caution should be taken in aspirating the pleural fluid, so that the subphrenic collection is not drained traumatically through this aspiration.

DR. BOYD (Closing).—In answer to the first discusser, I perhaps should have made the point that the biliary type of complication is much more serious from the standpoint of lung destruction. In our experience it has also been necessary to do a lobectomy for lung disease after bronchobiliary fistula. This has not been the case after the ordinary type of subphrenic infection with bronchial fistula.

If there were time one would like to have paid tribute to former Presidents of this Association and the present one, who have contributed much to our knowledge of subphrenic disease.

In answer to Dr. Carr, I might say that this group which we have treated, although quite large, is in a sense a selected series. Very few of these patients with subphrenic abscess have developed this complication following generalized peritonitis. In other words, they have been mostly of our own doing; that is, these abscesses have followed upper abdominal surgery which we have done ourselves. This may be the reason why there have been so many subphrenic rather than posterior subhepatic abscesses. In this series, Dr. Carr, we have not encountered any amebic abscesses, so that I am unable to answer your question.

THE EARLY ELECTIVE SURGICAL APPROACH TO THE TREATMENT OF TRAUMATIC HEMOTHORAX

Morris M. Culiner, M.D. (by invitation), Benson B. Roe, M.D., and Orville F. Grimes, M.D., San Francisco, Calif.

THE PRESENCE of blood in the pleural cavity represents a potential hazard and may prove to be a difficult problem to manage. The severity of the condition and its management depend upon several factors: the volume of blood and its rate of accumulation, the possibility of contamination, and the tendency of blood to undergo loculation and organization. The success of conservative management of hemothorax depends upon the absorptive capacity of the pelura and the ability to evacuate the pleural space by means of a needle or thoracostomy tube. Repeated futile attempts at treatment by conservative means, however, may only delay more definitive forms of therapy and increase their difficulties. The time required to absorb blood from the pleural space may serve to defeat conservatism in the management of hemothorax. If the blood becomes clotted, the possibility of evacuating blood through a tube or needle is lost and decortication becomes necessary to free the encapsulated lung. Frequently a more aggressive approach to the treatment of traumatic hemothorax not only answers the unknowns in the problem but permits them to be brought under control and materially reduces the over-all morbidity and duration of treatment.

Decortication of the lung is a well-accepted procedure in the treatment of clotted hemothorax. Most observers recommend that 4 to 6 weeks should elapse before elective decortication is attempted. This implies that the conservative approach to the evacuation of blood from the pleural space has failed. In civilian practice, when delays in evaluation can be kept at a minimum, the failure of conservative treatment should be recognized within several days. In these instances the thorax can and should be evacuated completely by early elective thoracotomy.

In order to assess our experience in the management of traumatic hemothorax and to justify early elective surgical intervention in certain patients, 43 case histories were reviewed. Most of these patients were cared for at the San Francisco General Hospital. This study extends over a period of 7 years, 1951

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to 1958. Of the 43 patients, 27 were treated with needle aspiration or tube drainage, 10 with early thoracotomy, 4 with delayed decortication, and 3 with rib resection for empyema. The following selected case histories illustrate some of the problems which occurred in the management of these patients.

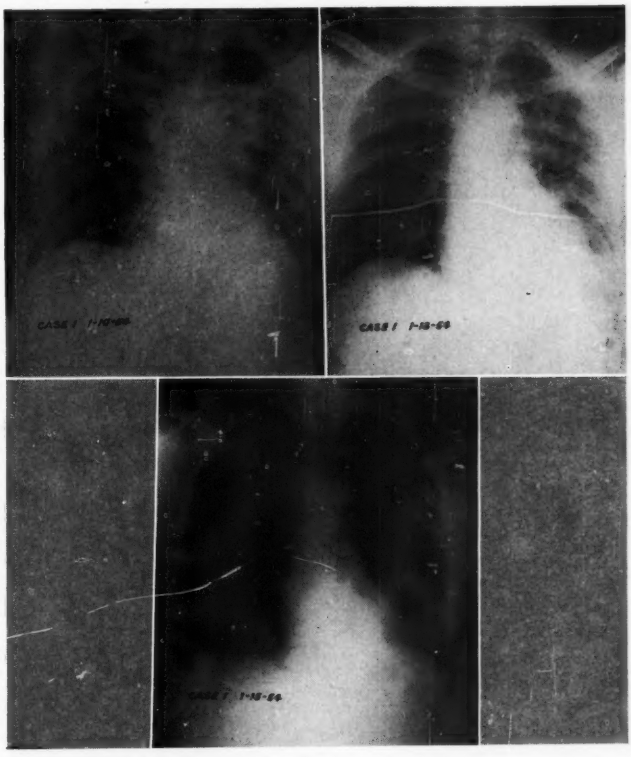


Fig. 1.—Case 1. *A*, The roentgenogram taken on admission shows haziness of the left hemithorax, due to hemopneumothorax.

B, The roentgenogram shows partial clearing following aspiration of 1,300 ml. of blood by the tubal thoracostomy and thoracenteses.

C, This film taken 5 days after trauma demonstrates a satisfactorily clear pleural space.

CASE REPORTS

CASE 1.—A 36-year-old Negro man sustained a stab wound from a penknife in the second left anterior intercostal space on Jan. 10, 1954. He was admitted to the hospital shortly after injury. A portable supine roentgenogram showed opacification of the left hemithorax (Fig. 1, *A*). Thoracentesis yielded 1,000 ml. of air. Tube thoracostomy was established 6 hours after the injury and suction was applied to the tube. Fifteen hundred milliliters of bloody fluid was evacuated (Fig. 1, *B*). Two transfusions of whole blood were administered. The thoracostomy tube was removed on the following day since it no longer functioned properly. During the next 4 days, needle thoracenteses were performed and a total of 300 ml. of bloody fluid was removed. A roentgenogram obtained on Jan. 15, 1954, 5 days after injury, showed the left hemithorax to be clear and lung expansion to be complete.

Comment: This case history illustrates the well-accepted concept that prompt tube thoracostomy serves to evacuate blood satisfactorily from the pleural space in most instances. The majority of the patients in this series were successfully managed by conservative means.

CASE 2.—A 38-year-old Negro man was stabbed in the third anterior right intercostal space and was admitted to the hospital within 30 minutes of injury. Although needle thoracentesis yielded 1,700 ml. of bloody fluid, evacuation of the pleural space was incomplete.

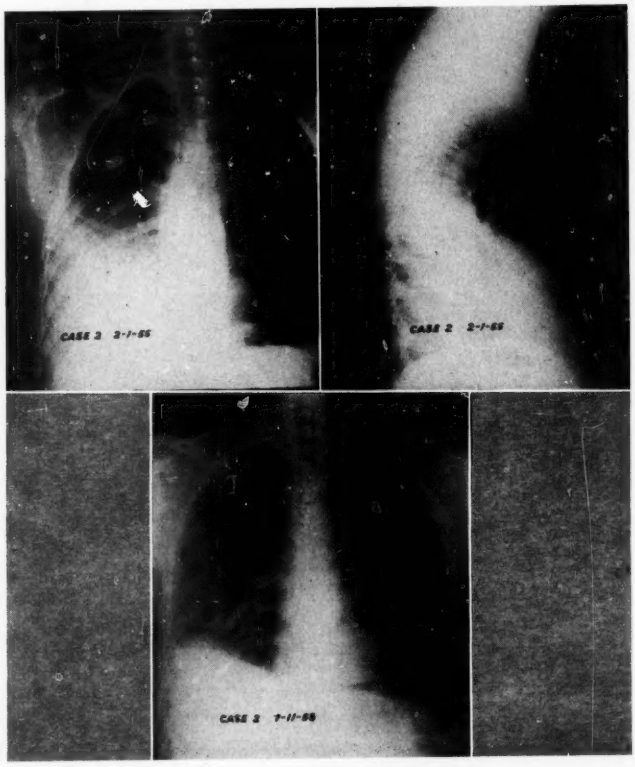


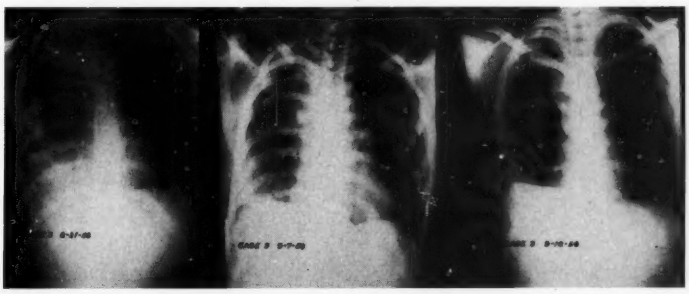
Fig. 2.—Case 2. *A* and *B*, Anteroposterior and lateral films taken 8 days after injury show a clotted hemothorax despite repeated early thoracenteses.
C, This roentgenogram demonstrates residual fibrotic organization of the hemothorax, 18 months after injury. Note the elevated and adherent right diaphragm.

A thoracostomy tube was inserted shortly after admission and was attached to 15 cm. of water suction. In spite of this therapy a considerable volume of blood remained intrapleurally. Thoracenteses were performed repeatedly in an attempt to obtain more complete evacuation of the pleural cavity. Very little bloody fluid could be aspirated by needle. Roentgenograms obtained on Feb. 1, 1955, 8 days following trauma, showed a large amount of blood loculated posteriorly in the right hemithorax (Fig. 2, *A* and *B*). At this time the patient signed out against medical advice. On July 11, 1956, 18 months after the stab wound was incurred, the patient was re-admitted to the hospital for treatment of a different

disease. A posteroanterior roentgenogram of the chest obtained at that time (Fig. 2, *C*) showed relatively good lung re-expansion. However, blunting and elevation of the diaphragm at the base of the right chest were present.

Comment: Although needle thoracenteses and tube thoracostomy on the day of injury resulted in evacuation of 1,700 ml. of bloody fluid, a large pocket of loculated blood clot still remained in the pleural space. After the tube was removed no effective treatment was possible. This patient spontaneously resorbed and organized most of the residual blood during the next 18 months but was left with a basal fibrothorax.

It is impossible to state whether a significant degree of respiratory reserve was lost in this patient since no specific studies were obtained. However, fluoroscopic observations of other patients with similar radiologic findings of blunting and elevation of the costophrenic angle have demonstrated markedly restricted ipsilateral diaphragmatic excursions. The clinical effects of this varied and depended upon other factors, such as the presence of parenchymal disease and the basic physiologic requirements of the patient's occupation.



A. *B.* *C.*

Fig. 3.—Case 3. *A*, Postoperative roentgenograms show hemopneumothorax with collapse of the right lung and the contracted hemithorax.
B and *C*, Roentgenograms taken 2 days and 13 days after decortication show that the right lung has re-expanded and the right hemithorax is quite clear.

CASE 3.—A 14-year-old Negro boy underwent exploratory thoracotomy to determine the nature of a right paramediastinal mass. Biopsy of the nodal mediastinal mass at that time revealed a tuberculous granuloma. Postoperatively the lung remained partially collapsed (Fig. 3, *A*). The patient was transferred to the San Francisco General Hospital on Aug. 21, 1956, 12 days after exploration. On admission to the hospital, examination showed markedly restricted respiratory excursions on the right. Pulmonary function studies indicated a moderate decrease in pulmonary reserve.

Roentgenograms and the aspiration of bloody fluid demonstrated a post-thoracotomy hemopneumothorax with partial collapse of the right lung. A thoracostomy tube was inserted into the pleural space on Aug. 24, 1956, and negative-pressure suction of 150 cm. of water was applied. Only 400 ml. of bloody fluid were evacuated during the next 30-hour period. Roentgenograms before and after tube thoracostomy revealed that no significant re-expansion of the lung had resulted. Decortication was believed to be advisable. On Sept. 5, 1956 (4 weeks after the initial traumatic hemopneumothorax) decortication was carried out. The patient did well postoperatively (Fig. 3, *B* and *C*). Intermittent positive pressure was administered frequently. Breathing exercises were administered and the patient showed a striking objective increase in respiratory function. The ultimate lung expansion was excellent and later respiratory function studies indicated a marked increase in reserve.

Comment: In this case, hemopneumothorax of 2 weeks' duration was first treated by tube thoracostomy and high suction. This treatment proved to be ineffectual in obtaining re-expansion of the lung and evacuation of pleural blood. Decortication was carried out 4 weeks after the initial hemopneumothorax. Decortication produced a good functional result.

CASE 4.—A 31-year-old Negro man was stabbed in the right upper anterior part of the chest on June 8, 1956. He was in shock when admitted to the hospital but his condition was stabilized by the administration of whole blood and plasma. He coughed up blood at this time, which suggested that the lung had been penetrated. A thoracostomy tube was inserted

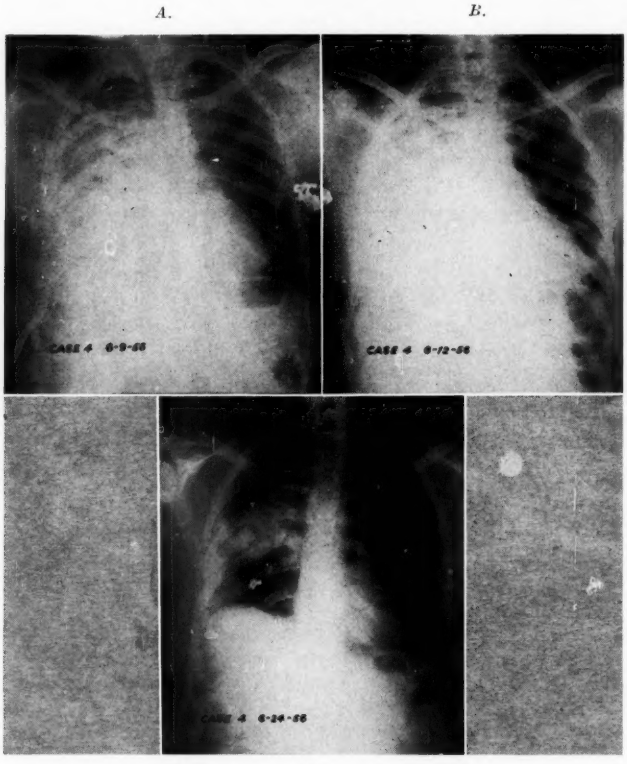


Fig. 4.—Case 4. *A*, A roentgenogram taken 1 day after injury shows the thoracotomy tube in the pleural space which is filled with clotted blood.

B, The residual hemothorax on the fourth day following injury is evident.

C, This film taken 12 days after early elective thoracotomy demonstrates the parenchymal hematoma and relatively clear pleural space.

immediately and suction was applied (Fig. 4, *A*). Only 500 ml. of blood was aspirated through the thoracostomy tube during the next 2 days. Roentgenograms taken 4 days after the initial trauma showed that a massive hemothorax was still present (Fig. 4, *B*). The patient was in good condition and showed no evidence of continued intrathoracic bleeding.

On the fourth day after injury an elective thoracotomy was performed. Approximately 1,500 ml. of clotted blood was removed. The pulmonary parenchyma had been lacerated by the stab wound but re-expansion of the lung was immediate and complete. No technical

difficulty was encountered in the removal of the blood clot. Films taken on the first postoperative day and on the twelfth postoperative day showed excellent lung expansion. The area of injury to the lung appeared as a hematoma in the mid-lung field (Fig. 4, *C*). The patient's further postoperative course was uneventful.

Comment: Conservative management was attempted unsuccessfully. The failure of tube thoracostomy was recognized early. At this point therapy might

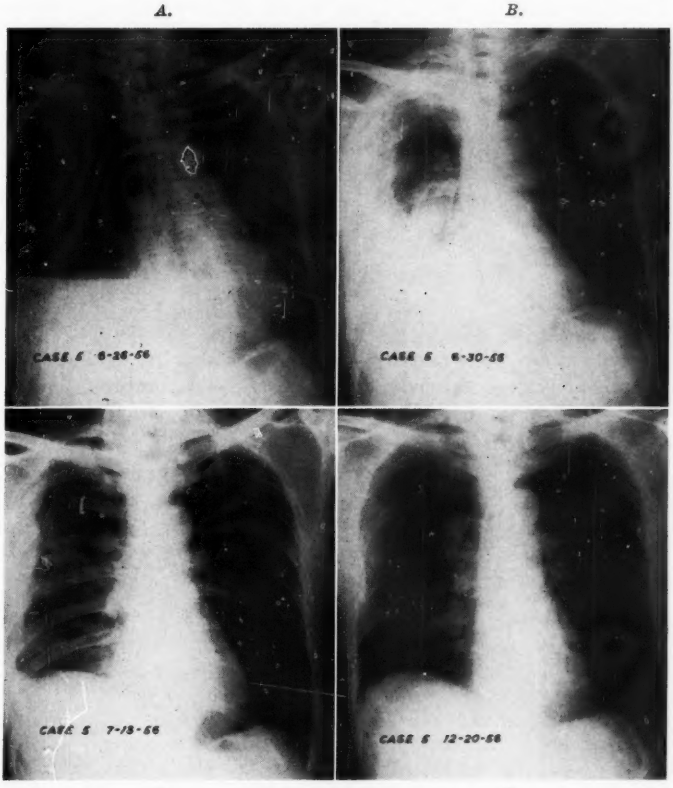


Fig. 5.—Case 5. *A*, This film taken on the day of admission shows massive spontaneous hemopneumothorax resulting in mediastinal displacement to the contralateral side.

B, Tubal thoracostomy produced an incomplete evacuation of the blood. Marked residual pulmonary collapse is shown.

C, A film taken 10 days following early elective thoracotomy for clotted hemothorax shows a relatively clear pleural space.

D, The follow-up roentgenogram demonstrates excellent lung expansion 5 months following thoracotomy.

have been directed along two other courses than that elected. On the one hand, the catheter might have been removed and the ability of the pleura to absorb the fluid then relied upon. On the other hand, a deliberate waiting period of 4 to 6 weeks could have preceded decortication. In each of these two choices the risk of failure and of complications must be recognized. Beyond that, such therapy prolongs hospitalization. After early elective thoracotomy this patient was home on the twenty-fourth day after injury.

CASE 5.—A 59-year-old Chinese man was admitted to the San Francisco General Hospital on June 26, 1956, with a history of tightness of the chest and shortness of breath for the previous 4 days. There was no history of injury. The initial roentgenogram showed an extensive right hemopneumothorax with mediastinal shift to the left and adhesions at the right apex (Fig. 5, *A*). The apical adhesions suggested that spontaneous rupture of the lung had occurred at this site.

Needle thoracentesis on the day of entry yielded 2,000 ml. of bloody fluid and 700 ml. of air. A follow-up roentgenographic study showed incomplete evacuation of the hemopneumothorax. Consequently on June 28, 1956, tube thoracostomy was performed but this also failed to evacuate the hemothorax although all air was removed and improved lung expansion followed. The tube was removed on June 30, the fourth hospital day, because its use had not resulted in adequate improvement. Elective thoracotomy was performed on July 3, 1956, 7 days after admission and approximately 11 days after the onset of hemopneumothorax. Thirteen hundred milliliters of fluid and clotted blood was removed. Satisfactory re-expansion of the lung was immediately obtained. An apical adhesion to which clotted blood had become adherent appeared to be the source of bleeding as postulated preoperatively. The operation was not technically difficult. The patient was discharged on the seventeenth hospital day, 21 days after onset of the hemopneumothorax. Roentgenograms at this time showed good lung expansion (Fig. 5, *C*). Roentgenographic studies 5 months later showed an excellent result (Fig. 5, *D*).

Comment: This patient with hemopneumothorax was initially treated conservatively with tube thoracostomy. When this therapy proved ineffective, elective thoracotomy was carried out on the seventh hospital day. On the seventeeth hospital day, 10 days postoperatively, the patient was discharged with a totally satisfactory result.

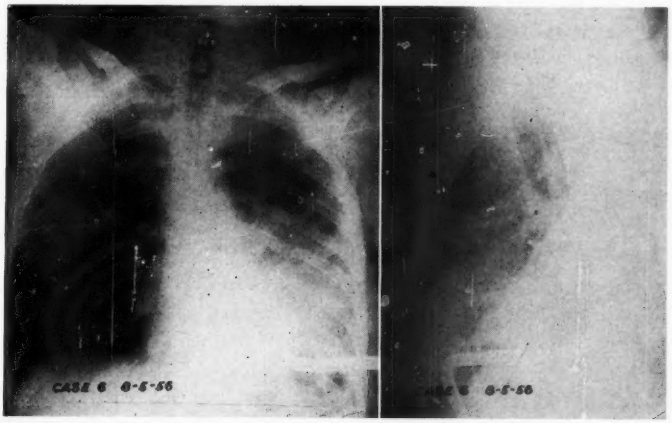
CASE 6.—A 31-year-old white man was stabbed in the fourth left intercostal space on Aug. 4, 1956. Needle thoracentesis was performed shortly after arrival at the hospital and within a few hours following injury. Since adequate improvement was not obtained a thoracostomy tube was inserted and suction was applied. Despite the early insertion of the tube into the pocket of pleural blood, as demonstrated on the roentgenograms (Fig. 6, *A* and *B*), only 400 ml. of bloody fluid was aspirated. Most of the blood had clotted and remained intrapleurally. The patient's condition stabilized within 12 hours after injury and after 2 units of whole blood had been administered.

Tube drainage with suction yielded only 200 ml. of blood during the next 18 hours. Repeated roentgenograms again showed a large left posteroinferior density of the hemithorax. Fifty-six hours after injury, an elective thoracotomy was performed. Approximately 1,300 ml. of blood, clot, and fluid was removed without difficulty. The lower lobe of the left lung was found to be completely compressed by the blood and clot but it re-expanded readily. The patient was discharged with excellent lung aeration on Aug. 19, 1956, the fifteenth day after injury (Fig. 6, *C* and *D*).

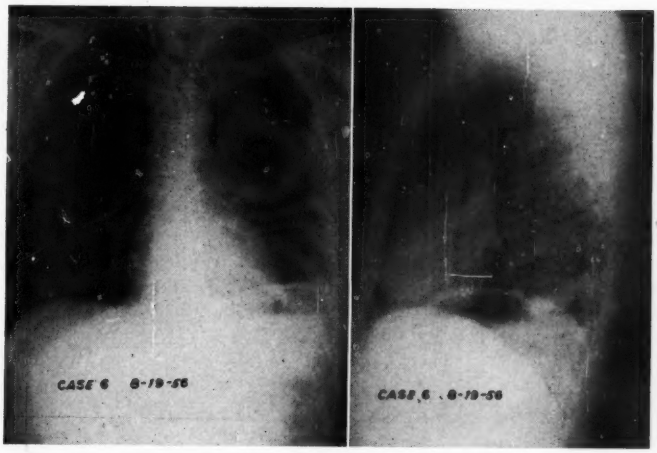
Comment: Although this patient was treated for hemothorax by tube thoracostomy within several hours of injury, the hemithorax was inadequately evacuated by this method. Elective thoracotomy with evacuation of the clotted blood was carried out 56 hours after injury. The functional results were excellent and the degree and duration of illness and disability were minimized, in addition to eliminating the possible complications of infection of a clotted hemothorax.

CASE 7.—A 40-year-old white woman was admitted to the hospital on Nov. 18, 1955, a few minutes after being in an automobile accident. Roentgenograms of the chest showed fractures of the eighth, ninth, and tenth ribs on the right side (Fig. 7, *A*). These initial films showed no other abnormalities and specifically did not reveal evidence of fluid in the right pleural space. Follow-up roentgenographic studies 3 days after the injury showed a

large amount of fluid within the right pleural space (Fig. 7, *B*). Thoracentesis yielded 800 ml. of bloody serous fluid. Subsequently 2,000 ml. of serosanguineous fluid formed but satisfactory expansion of the lung was obtained by simple needle evacuation (Fig. 7, *C* and *D*).



A. *B.*



C. *D.*

Fig. 6.—Case 6. *A* and *B*, The thoracostomy tube is seen in the posteriorly loculated hemothorax. Only the lateral view gives an accurate indication of the extent of the hemothorax.

C and *D*, The roentgenograms taken on the fifteenth day after injury and 12 days after early elective thoracotomy show satisfactory clearing and lung expansion.

Comment: This case is of interest because it demonstrated the phenomenon of accumulation of fluid or blood several hours or days after injury and after an initially clear pleural space had been shown roentgenographically. This suggests the necessity of follow-up films and careful observation even after early negative radiologic findings.

CASE 8.—A 25-year-old Negro man was stabbed through the left fourth interspace anteriorly on Oct. 23, 1958, and was admitted to the hospital within 1 hour following injury. At that time he was dyspneic and had a pulse rate of 120. Respirations were 30 per minute and blood pressure was 100/80 mm. Hg.

A roentgenogram of the chest revealed a left hemopneumothorax (Fig. 8, *A*). Two intercostal catheters were inserted and connected to continuous suction. During the next 6 hours, 2,700 ml. of bloody fluid was evacuated from the left pleural space and 2,500 ml. of

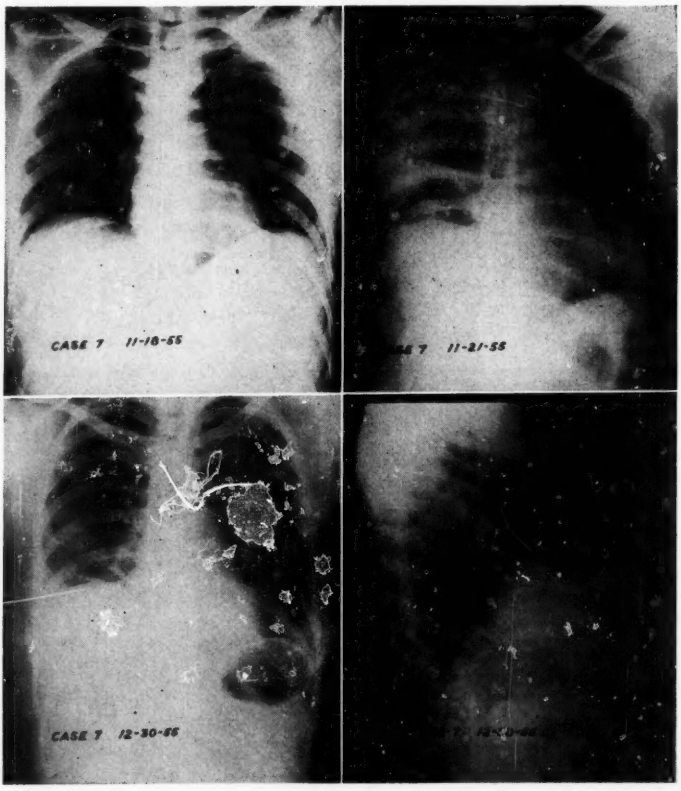


Fig. 7.—Case 7. *A*, A clear right hemithorax is seen shortly after injury. *B*, A delayed accumulation of serosanguineous fluid is demonstrated 3 days after injury. *C* and *D*, Thoracenteses resulted in a clear hemithorax following delayed hemothorax formation.

whole blood was administered intravenously. The thoracostomy tubes continued to drain about 300 ml. of blood per hour. Eight hours after injury, a thoracotomy was done. At operation, 1,000 ml. of blood clot and 800 ml. of bloody fluid were removed from the left pleural space. The only active bleeding was from the intercostal muscles around the stab wound. A short laceration of the lung was present but was not bleeding at the time of thoracotomy.

All bleeding was controlled with suture ligatures. Expansion of the lung was readily obtained and maintained (Fig. 8, *B*). Pleural fluid was demonstrated radiologically on the

sixth postoperative day. Thoracentesis yielded 1,200 ml. of blood-tinged serous fluid and the hemithorax was completely cleared by this (Fig. 8, *C*). The patient's postoperative course was otherwise uneventful and he was discharged on the tenth postoperative day.

Comment: This patient demonstrated continued and significant bleeding after early tube thoracostomy and evacuation of 2,700 ml. of blood were carried out. Thoracotomy was thought to be necessary and was performed 8 hours after the patient was admitted to the hospital. At thoracotomy there was 1,900 ml. of blood and clot within the pleural space despite the presence of 2 tubes to which suction had been constantly applied.

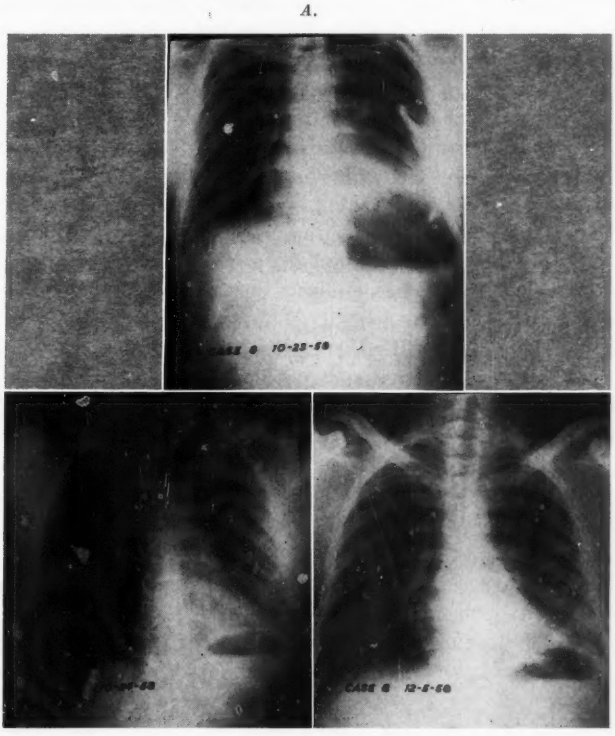


Fig. 8.—Case 8. *A*, The roentgenogram demonstrates the post-traumatic hemopneumothorax.
B, This film was taken 1 day following trauma and thoracotomy. A total of 4,500 ml. of blood and clot had been evacuated by tube and thoracotomy.
C, The follow-up roentgenogram shows satisfactory clearing.

DISCUSSION

The care of patients with early traumatic hemothorax cannot be rigidly standardized. A basic plan of treatment can be formulated and this can be appropriately altered as new developments arise. The results of early therapy will necessarily influence the subsequent steps in the management. In traumatic hemothorax the hemorrhage usually ceases spontaneously and the patient's

condition becomes stable with fluid and blood replacement. In these patients thoracotomy is not necessary as a lifesaving measure. The problems of paramount importance are the preservation of maximum pulmonary function and the prevention of sepsis. Whether a significant decrease in pulmonary reserve occurs as a result of hemothorax depends upon the volume of clotted blood compressing the lung, the development of infection, and the production of a fibrous restriction of the respiratory movements. The resulting respiratory embarrassment also depends on the pre-existing pulmonary reserve.

The more complete the evacuation of blood from the pleural space the better is the functional result. The earlier the blood is removed the more complete is the evacuation, since later clotting and loculation will prevent emptying of the pleural space by conservative means.

In many patients with traumatic hemothorax of minor degree, intrapleural blood can be aspirated by needle within the first few hours following trauma. Needle thoracentesis carries only a minimal risk of pleural contamination unless it is repeated often. This technique also carries very little danger of pneumothorax due to puncture of the lung. Certain disadvantages, however, must be kept in mind; the small caliber of the needle does not permit rapid removal of blood; the needle easily becomes plugged and complete removal of blood from the pleural space is not often accomplished in this manner.

Should the amount of blood in the pleural cavity be at all significant, that is, sufficient to obscure the diaphragmatic leaf and costophrenic angle, tube thoracostomy should be used as soon as possible. The method of Betts and Lees[1] may be helpful. With this technique, the trocar used for insertion of the intercostal catheter remains briefly in place. The catheter is moved around all areas of the pleural space while constant suction is applied to it. After all possible blood has been evacuated, the trocar is removed. The catheter remains intrapleurally and is attached to suction to evacuate blood which remains and serous fluid which may continue to ooze into the pleural space.

Often, although the pleural cavity may have been cleared initially, reaccumulation of fluid may be noted after several days. This fluid may result from continued bleeding or from the exudative reaction of the pleural surfaces, not only to the initial trauma, but also to the blood which remains. When early roentgenograms show the pleural space to be devoid of fluid or if complete evacuation of the hemothorax has been carried out, follow-up roentgenograms should be obtained several days later to demonstrate whether re-accumulation has occurred.

The clotting of blood in the pleural cavity may occur within a few minutes or may be delayed for several days following injury. Often, despite the early use of needle thoracentesis and tube thoracostomy, large amounts of blood may remain clotted in the pleural space. Our experience with the use of proteolytic enzymatic agents has been disappointing. Use of these agents has frequently produced an increased volume of fluid which can be aspirated from the pleural space. It is quite likely that the increased fluid resulted largely from the exudative reaction of the pleural surfaces to the irritating agents.

The significant problem is the selection of patients in whom removal of clotted pleural blood by open thoracotomy is warranted. A review of the cases of several patients with hemothorax who were essentially untreated has indicated that resorption or fibrotic organization of blood and concomitant re-expansion of the lung may occur spontaneously. Lung re-expansion as indicated by roentgenograms may occur in the presence of a restrictive fibrotic pleural process which can significantly inhibit respiratory excursions. For this reason a state of lung expansion in itself does not indicate good ventilatory function. In problems such as these, pulmonary function studies, especially bronchospirometry, may provide the only objective determination of the decrease in pulmonary function produced by the fibrotic pleural process. Without similar studies recorded prior to injury even these studies have only limited significance.

The decision for surgical evacuation of blood must be based on a clinical assessment of the following factors: (1) the ventilatory excursions of the thoracic wall; and (2) roentgenographic demonstration by posteroanterior and lateral views of the pleural blood and its position. This assessment can be made as accurately within a few days as after 4 to 6 weeks. During this period, minimal resorption of clotted hemothorax occurs. During the same period, however, there can be resolution of pleural reaction and clearing of the roentgenogram. However, the radiologic picture of clotted hemothorax must be differentiated from that of pleural reaction and thickening. These conditions may resemble each other on posteroanterior roentgenograms. Lateral views, however, generally distinguish between the 2 entities since a clotted hemothorax frequently shows a postero-inferior triangular opacified area due to loculation. A diffuse circumferential opacification suggests pleural reaction. This differential point was emphasized by Tuttle.[7]

When it becomes apparent that conservative means of removing blood from the pleural cavity are inadequate and that optimum functional results require the evacuation of clotted blood, operation is carried out without delay. Early elective thoracotomy for decompression of the lung and evacuation of blood clot has been performed within periods ranging from a few hours to 17 days following the formation of hemothorax. These operations have been performed with no great technical difficulty and full lung re-expansion has been obtained as readily as when decortication has been employed 4 to 6 weeks after trauma.

Some patients show evidence of continuing severe intrapleural bleeding after initial evacuation of a hemothorax. These patients should be treated by early elective thoracotomy to control the source of bleeding and to evacuate the pleural space completely.

By performing a thoracotomy within a few hours or days after injury one can control the hemorrhage, evacuate all blood clots, permit and maintain immediate lung re-expansion and, in most instances, prevent the development of empyema. With adequate replacement of whole blood these patients are managed satisfactorily during operation. The source of bleeding is most frequently a lacerated intercostal vessel. In those cases where early elective

thoracotomy has been utilized, lung re-expansion has been easily obtained and the postoperative course has been quite uncomplicated. By immediate or early lung expansion with obliteration of the dead intrapleural space and removal of the excellent culture medium, empyema has been prevented in all cases. The economic factors of early operation and convalescence are quite important to the patient. No benefit is derived from a delay of 4 to 6 weeks from the time of onset of clotted hemothorax to the time of evacuation.

In this series of 43 patients treatment was as follows: (1) 27 patients were treated by needle thoracentesis with or without tube thoracostomy, (2) 10 patients were treated by early elective thoracotomy within a few hours or days following trauma, (3) 4 patients were treated by delayed decortication 4 to 6 weeks after injury, and (4) 2 patients were untreated until empyema occurred and then were treated by rib resection.

Of the 27 patients treated by needle thoracentesis with or without tube thoracostomy, 26 did well. One patient treated early in the series developed empyema. This patient had been ineffectively managed by repeated thoracenteses and instillation of antibiotics and proteolytic agents. Early thoracotomy with evacuation of blood clot would have been much more appropriate therapy in this case.

Of the 10 patients treated by early elective thoracotomy for hemothorax, 9 patients recovered promptly. The one death in the series occurred 12 hours after secondary thoracotomy for hemothorax. The primary sternum-splitting thoracotomy had been done to remove an anterior mediastinal neurilemmoma. A secondary thoracotomy was carried out 48 hours after the primary procedure. The cause of death was not definitely established. It was probable, however, that there was undue delay in carrying out the secondary thoracotomy to control the postoperative hemothorax.

The 4 patients treated by delayed decortication, at intervals of 4 to 6 weeks following trauma, had uneventful postoperative courses and satisfactory results. All of these patients came under our treatment at a late stage. The average period of hospitalization for the patients who underwent decortication was 48 days. In contrast with this, the average stay of patients treated by early elective thoracotomy was 19 days.

Three patients whose condition was complicated by infection were treated by rib resection and drainage.

In 1 patient, empyema developed immediately in a hemothorax after he had suffered a gunshot wound. The first sample of blood aspirated from the pleural space revealed the infecting organism. The second patient signed himself out of the hospital after several days only to return in 2 weeks with frank empyema. The empyema spaces in these patients were sufficiently small so that decortication was not believed to be warranted. The third patient developed a hemothorax following resection for coarctation. The hemothorax was treated by thoracenteses only, and the patient developed empyema 18 days afer the primary procedure. This patient should have had early elective thoracotomy rather than repeated ineffective thoracenteses. As a result of the ill-advised therapy with aspiration and instillation of antibiotics and proteolytic agents, he was

hospitalized for 2 months. All 3 of these patients were treated by rib resection and open drainage. Ultimately re-expansion of their lungs was satisfactory and the empyema cavities eventually healed.

SUMMARY

Selected case reports from a series of 43 patients with hemothorax are presented to demonstrate the problem and results associated with the various forms of treatment. A rationale is developed for selected early surgical intervention as the treatment of choice when conservative measures are unsatisfactory. Experience has indicated that the morbidity and mortality of thoracotomy are low and that the results are definitive and complete in removing the hemothorax. The hospitalization and disability periods are significantly shortened. Early elective thoracotomy is recommended for clotted hemothorax in preference to a deliberate delay for cortex formation.

REFERENCES

1. Betts, R. H., and Lees, W. M.: Military Thoracic Surgery in the Forward Area, J. THORACIC SURG. 15: 44-63, 1946.
2. Harken, D. E.: A Review of the Activities of the Thoracic Center for the III and IV Hospital Groups, 160th General Hospital European Theater of Operations, June 10, 1944, to Jan. 1, 1945, J. THORACIC SURG. 15: 31-43, 1946.
3. Jonas, A. F.: Massive Organizing Hemothorax; Operative Treatment, Its Technique, Indications and Pitfalls, Surgery 20: 168-173, 1946.
4. Samson, P. C., Burford, T. H., Brewer, L. A., and Burbank, B.: The Management of War Wounds of the Chest in a Base Center; the Role of Early Pulmonary Decortication, J. THORACIC SURG. 15: 1-30, 1946.
5. Samson, P. C., and Burford, T. H.: Total Pulmonary Decortication; Its Evolution and Present Concepts of Indications and Operative Technique, J. THORACIC SURG. 16: 127-145, 1957.
6. Seley, G. P., and Neuhof, H.: Pulmonary Decortication for Clotted Hemothorax Following Idiopathic Spontaneous Hemopneumothorax, J. THORACIC SURG. 21: 600-601, 1951.
7. Tuttle, W. M., Langston, H. T., and Crowley, R. T.: The Treatment of Organizing Hemothorax by Pulmonary Decortication, J. THORACIC SURG. 16: 117-126, 1947.
8. Tuttle, W. M., Langston, H. T., and Crowley, R. T.: The Treatment of Intrathoracic Wounds, Surg. Gynec. & Obst. 81: 158-168, 1945.
9. Maloney, J.: Conservative Management of Traumatic Hemothorax, Am. J. Surg., April, 1957.
10. French, S. W., and Rumer, G. F.: Limited Thoracotomy and Enzymatic Therapy in the Treatment of Early Clotted Hemothorax, U.S. Armed Forces M. J. 9: 949, 1958.

DISCUSSION

DR. CHARLES T. READ, Phoenix, Ariz.—I hesitate to get up before this group again to discuss streptokinase, but I didn't hear the word mentioned in the paper that was just delivered. I heard proteolytic enzyme mentioned one time. I hope it was not one of the competitive drugs.

I am sorry that I happened to be the man who came to this meeting, but just happened to have no slides with me. But I venture to say that in my own experience with post-resection problems, which I have shown before this group, they look much worse than some of these cases that were shown, and I feel that enough emphasis has not been placed upon this particular maneuver. If the authors did not use this procedure, I think they have passed up a good portion of the armamentarium offered in taking care of these patients' problems, because with the use of closed thoracotomy one has the advantage of using enzymes quite frequently until the chest is evacuated, and in many instances very good results can be obtained.

If these gentlemen have used this, I would like to hear more about it. If they haven't, I would like to remind them of the work that has been carried on previously.

DR. JOSEPH A. WEINBERG, Long Beach, Calif.—I interpret this paper, which I certainly enjoyed, as advocating an earlier recognition of the patient who will need more than simple, watchful treatment, aspiration, or possible tube thoracotomy.

I would like to point out that the pleural surfaces have remarkable absorptive power for clearing the blood from the hemothorax, and that I trust that the remarks that have been made here will not be interpreted as advocating open thoracotomy with evacuation of blood and clots in all patients. I am sure that the authors do not mean that, since more than half of their patients were treated by more conservative means. Operations are not without danger and there should be clear indications for their use.

I also would advocate conservatism in the waiting period while it is being determined whether or not the patient will need an open thoracotomy with evacuation of clot and possible decortication. Frequent aspirations may be the very thing that makes open operation necessary at a later time because of the introduction of contaminating bacteria with subsequent infection.

DR. ROBERT K. BROWN, Denver, Colo.—I agree 100 per cent with the authors when they say that these things should be cleaned out and normal physiology restored. I thought Dr. Weinberg would make the point I want to make, but he didn't. I think it is very important in the thoracotomy for this situation to do it through an intercostal incision and leave an intact 12-rib hemithorax for the best and most efficient possible restoration of ventilatory function after operation.

DR. JAMES V. MALONEY, JR., Los Angeles, Calif.—Two years ago we published our own results in a group of 40 cases of traumatic hemothorax with very similar results. We interpreted our experience as indicating that surgical decortication should be used less often and later, rather than more often and earlier.

There has been a general trend toward the surgical treatment of clotted hemothorax since the very fine and classic work of Dr. Samson. We became worried, however, that this trend was being carried too far. In self-evaluation I returned to Baltimore, where I had treated a large number of these patients over a period of 8 or 9 years. I was able to find follow-up chest films (taken for incidental diseases, tuberculosis surveys, et cetera) in 40 cases. Thirty of the hemothoraces were due to gunshot and stab wounds and ten were due to operation. We were amazed to find that on the incidental chest films taken from 3 months to 7 years later there was not a single instance of clotted hemothorax in which there was any detectable evidence of chest abnormality (with the exception of retained missiles) in any of the 30 cases due to stab wounds or gunshot wounds. Postoperative hemothorax was a more difficult problem and required surgical decortication in 3 cases and enzymatic decortication in 5. Empyema was encountered with enzyme therapy and, like Dr. Culiner, we had one death in the operative group.

The technical ease of decortication and the restoration of the chest film to normal give this operation a special appeal to the esthetic sense of the surgeon. But the major question is whether or not the operation is necessary. I am sure there is merit to both the conservative treatment that we recommend, and also to the idea that Dr. Culiner has presented today. I don't know which point of view is correct, but it is appropriate that the subject be brought up and re-evaluated so that we can obtain a definitive answer to the problem in the next several years.

DR. EDOUARD D. GAGNON, Montreal, Quebec.—We, too, have been interested in early thoracotomy when indicated in hemothorax. I would like to refer specifically to 3 such cases where there was traumatic hemopneumothorax because, in these cases, without thoracotomy (the two first ones on the day of injury and the third one 5 days after injury, when first seen by us) there might have been very severe or dire consequences, because the source of hemorrhage was a "split" lung.

The most interesting thing to me was that in the first case the right upper lobe had been split wide open by this closed injury to the chest, as if with a meat cleaver, and the bleeding was coming from the lobar veins, whereas the air was leaking out of multiple bronchioles plus an additional tear to the lobar bronchus. This patient was treated by lobectomy.

In the second case, the left upper lobe and left lower lobe were split open as if with a knife on their posterior aspect; the same pathology was present in the third case, but on the right side. When the lung was fitted back into the posterior gutter it seemed to me, in these 2 cases, that the injury had been produced by the anterior forward edge of the vertebral column, and the lobes had been split open as if they had been cut with a knife, or with a meat cleaver.

The second case required resection because the lung was very damaged. In the third case we were able to suture the sliced parenchyma and the patient made an uneventful recovery.

It is, I think, worth remembering such possibilities in closed injuries to the chest. If the trauma has been severe enough to split the lung and occasion hemorrhage, early thoracotomy, or earlier thoracotomy, in these cases might save a few lives.

Maybe the reason we surgeons have not recognized this condition more often is that a great majority of these cases probably end up in the civic morgues.

DR. PAUL C. SAMSON, Lafayette, Calif.—This paper by Drs. Culiner, Roe and Grimes has interested me very much, and I am sure they will not be too hard on me if I disagree with them somewhat.

I think no one will argue for a moment that there are cases in which early thoracotomy in traumatic hemothorax is a very wise procedure. They indicated the fact, as did Dr. Gagnon, that in cases of continued bleeding thoracotomy is certainly necessary.

On the other hand, in the cases under discussion, and I would like to limit my remarks to those patients in whom we wish to preserve lung function, I doubt very much if 25 per cent of the time we need to do a thoracotomy to achieve the result desired. You will note that the essayists operated on 25 per cent of their patients.

Their series is small, and I presume that this is their entire experience, at least since they started doing thoracotomy, but I would like to remind them that we have cared for hemothoraces for a number of years. Perhaps blood clots more frequently in San Francisco than it does in Oakland, but as we have reviewed briefly our series of cases at the County Hospital, where we see 20 to 30 traumatic hemothoraces a year, I cannot find evidence of having operated in more than 1 out of 12 or 15 cases. In other words, it is not the fact that we never operate on hemothoraces, but we don't operate as much as is done in San Francisco.

I hope it doesn't date me if I remind you that even today thoracotomy carries at least a statistical risk, and that we should not operate any more than is absolutely necessary.

My only advice in closing is that I think they should use the needle more and the scalpel less.

DR. LAURENCE MISCALL, New York, N. Y.—I would like to enthusiastically support Dr. Samson.

During the last war, when the late Dr. Elliot Cutler was being inducted into the Royal College of Surgeons, he asked me to look over some of the data from the hospitals which were not thoracic centers. I reviewed 200 consecutive cases of hemothorax which were treated initially in hospitals other than thoracic centers where supposedly decortication and other definitive chest operations were to be done. I found that when repeated aspiration or tube drainage was used with thoracic exercise as indicated in the field, evacuation and station hospitals, about 75 per cent of these 200 patients recovered completely and never had to undergo any surgery for hemothorax.

I think there is another point of importance. I do not believe that one can transpose all the war experience into civil life. Many of our military patients had to come to thoracic

centers for secondary operation for hemothorax because of other factors. Many had retained large foreign bodies often with infection. Clotted or infected hemothoraces requiring drainage or decortication were therefore more common than might be expected in civil practice.

I would like to make one more observation since I noted that tubes were placed in some chests quite early. Frequently have I seen a too enthusiastic assistant resident treat a stab wound of the chest by immediate tube drainage and to his utter surprise found out in a short time that he is sucking out from the patient more blood than he can easily replace.

DR. JOSEPH GORDON, Albuquerque, N. Mex.—Now that Dr. Samson has opened the wound, I thought that I would like to add just a little bit of salt into it by bringing these factors to your attention. Commonly, when an injury to the chest occurs and hemothorax is present, there are two other factors; that is, most often there is contusion of the lung, in addition to which the body defense mechanism puts the diaphragm and the chest at rest. The diaphragm elevates itself to a "mean position" in the respiratory phase, and the rib cage resists movement to protect itself from pain caused by the injury.

Now, to swing this treatment over a little bit more to the more conservative side, one of the features that we should recognize, and Dr. Weinberg indicated that the pleurae have a large absorptive ability, is that it is necessary to return this body to a physiologic function as quickly as possible. If you don't have to put a needle in the chest because of respiratory distress it is best to exercise these patients. Make the patients move their chests, make them move their diaphragms; they will more rapidly return to physiologic function and heal the injury which has occurred while absorbing the fluid.

DR. JOSEPH L. LUCIDO, St. Louis, Mo.—We have had an experience with 300 traumatic chest injuries at the St. Louis County Hospital in St. Louis, Missouri, during the past 10 years. We have followed the points emphasized by Doctors Tom Burford, Paul Samson and associates in their papers published after the Second World War.

You will recall that approximately 15 per cent of all the hemothoraces which they reported became clotted and 85 per cent remained liquid early in the course of the disease. We have, therefore, used two, three or even four large water-seal tubes early in the course of the disease of these patients. If maximum expansion and evacuation has not been obtained in 72 hours we have then resorted to massive use of Tryptar or Varidase, and I am mentioning the names. We have used Tryptar or Varidase massively 7 or more days before resorting to thoracotomy.

If the above has not been successful we have then, as stated, resorted to thoracotomy and decortication. Exploratory thoracotomy has been used during the first 48 hours in individuals with massive uncontrollable hemorrhage, or other severe associated injuries of an emergent nature. I recall that last year we reported 25 instances in which we explored individuals who had ruptured diaphragms.

Our thoracotomy percentage has been less than 5 per cent in the individuals whom we have treated in this manner.

DR. CULINER (Closing).—I realized that bringing up this subject was somewhat like bringing coals to Newcastle, and I thought it might precipitate a little bit of discussion, not this deluge.

I would like to indicate that we have heard of streptokinase, streptodornase, and Tryptar and have used them extensively earlier.

[Slide] We feel that certainly with the use of proteolytic agents one can always aspirate a great deal of fluid following their instillation, whether it is in 6 hours or 24 hours. My own feeling is that the fluid that one aspirates following their use is the fluid that one causes by their use.

This particular patient illustrates one whom we treated with these magic drugs, and this film of September 19 shows the hemothorax present in the right lower portion of the pleural space. On this day streptokinase and streptodornase were instilled, and on the following day we had this degree of pulmonary collapse and formation of jelly-like material, which

we later evacuated by thoracotomy. Our own feeling is that with the use of streptokinase and streptodornase we have not been as successful as many of the papers on the subject would indicate, and therefore we use it less, probably, than other services.

We are also fully cognizant of the fact that the absorptive capacity of the pleural surfaces is quite marked. We haven't been as fortunate as Dr. Maloney in getting follow-up films. We have tried very hard to have patients treated in San Francisco County Hospital return for follow-up films. It has been difficult for us. In those few of whom we have been able to get follow-up films, in patients who refused therapy and who had a significant degree of hemothorax initially, we have found that many of them showed quite a marked pleuritic reaction with elevation of the diaphragm. Fluoroscopic examination of these has revealed inhibited respiratory excursion of the diaphragm and the lower hemithorax.

Perhaps the fog in San Francisco clots the blood more readily than it does in Oakland. Our 27 cases handled in that manner were quite satisfactory to us, but in 10 cases we felt it necessary to do early elective thoracotomy.

THE SURGICAL TREATMENT OF PEPTIC ESOPHAGITIS

Frederick S. Cross, M.D., George V. Smith, Jr., M.D. (by invitation), and Earle B. Kay, M.D., Cleveland, Ohio

INTRODUCTION

WHAT is presently referred to as peptic or reflux esophagitis was first described by Quinke in 1879[21] and by MacKenzie in 1884.[17] It has gradually evolved as a definite clinical entity through the work of numerous clinical investigators[1, 4, 5] whose observations have been substantiated by laboratory experimentation.[3, 13]

When peptic esophagitis becomes so severe as to result in intractable pain, bleeding, stenosis, or perforation, medical therapy must be abandoned and some form of surgical therapy considered. As to what constitutes proper surgical treatment for these devastating complications of esophagitis has been a stimulus to surgical investigators for a number of years. A variety of procedures have been offered, ranging from phrenic nerve paralysis,[20] gastric resection,[18] esophagogastrectomy,[24] and hiatal hernia repair,[11, 16] to rather complicated plastic procedures on the esophagogastric junction.[2, 12, 22]

It is difficult to draw categorical conclusions as to the efficacy of any given procedure from reviewing the literature since, in many instances, conclusions are based on an experience obtained from the analysis of relatively few patients. In a recent study of 66 patients with reflux esophagitis, seven different operative procedures, or combinations thereof, were performed on 60 patients.[23] No single combination was done more than seven times. In another similar series of 60 patients with reflux esophagitis, there were no fewer than eight types of operations done.[14] The largest experience was with a combination of gastric resection and hiatal hernia repair performed on 14 patients.

Perhaps part of the confusion as to proper therapy, medical as well as surgical, but more particularly surgical, lies in the fact that everyone may not be talking about the same thing when he refers to esophagitis. Moersch and his associates[19] have aptly stated that esophagitis is not a single disease entity, but rather a group of conditions of related origin. Esophagitis secondary to prolonged vomiting differs markedly from that resulting from the nighttime

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puddling of gastric secretion after passage through an incompetent hiatus. In addition, Barrett[5] adds a third type occurring in an esophagus lined with columnar mucosa.

Further confusion as to the proper operation for reflux esophagitis results from the presence of conditions existing in combination with the esophagitis. For instance, a hiatal hernia is frequently present, there may be an associated gastric or duodenal ulcer, or the esophagitis might be the result of surgical destruction of the cardiac sphincter mechanism, as in esophagogastrectomy. There is no disagreement that the damaging effects of acid-peptic juice on the vulnerable esophageal mucosa must be curtailed if esophagitis is to be cured, but how to weigh the significance of these contributing factors in the light of adequate therapy poses a problem, and leads to the variations in opinion existing today.

The purpose of the present report is to review critically a group of patients with reference to the degree of existing esophagitis and to the presence of contributing etiological factors; to re-emphasize the role of hiatal hernia in the production of peptic esophagitis; and to propose reconstruction of the cardiac sphincter mechanism, as in hiatal hernia repair, as the logical treatment of this disease.

DEFINITION OF TERMS

For reasons previously stated, it is extremely important to define terms as accurately as possible when discussing the treatment of reflux esophagitis. For the purposes of this study, esophagitis has been categorized primarily as that existing with a hiatal hernia, and that in which no demonstrable hiatal hernia was present. However, the fact that no hiatal hernia was demonstrated does not necessarily preclude the existence of one. The degree of esophagitis has been graded into severe, moderate, and minimal. It is somewhat difficult to categorize esophagitis in this manner with extreme accuracy; but, inasmuch as it is possible, this was done on the basis of x-ray and esophagoscopic findings as outlined in Table I. Any patient with significant bleeding, stenosis, or perforation of the esophagus was listed as having severe esophagitis. Dysphagia alone

TABLE I. CLASSIFICATION OF REFLUX ESOPHAGITIS

TYPE	ESOPHAGOSCOPY	X-RAY
Mild	Reddening and edema of mucosa Prominent longitudinal folds Hyperplastic mucosal changes Reflux of gastric juice	Reducible concentric hernia Peristaltic abnormalities Reflux of barium
Moderate	Linear erosions Spasm and edema Friable, easily bleeding mucosa Reflux of gastric juice	Reducible concentric hernia Reflux of barium Spasm or narrowing of terminal esophagus Peristaltic abnormalities Irregularity of mucous membrane of distal esophagus
Severe	Ulceration with bleeding Granular denuded areas Marked edema and spasm Stenosis Reflux of gastric juice	Irreducible concentric hernia Shortening of terminal esophagus Ulceration of mucous membrane Reflux of barium Stricture of terminal esophagus

was not considered a symptom suggesting stenosis, since dysphagia can result from associated neuromuscular changes in the esophagus as well as from stenosis.[9] In most instances, the hernia was of the concentric or direct type in which the acute angle of entry of the esophagus into the stomach is lost. If the hernia was irreducible, with associated narrowing or shortening of the esophagus, the patient was placed in the severe category (Fig. 1); whereas,

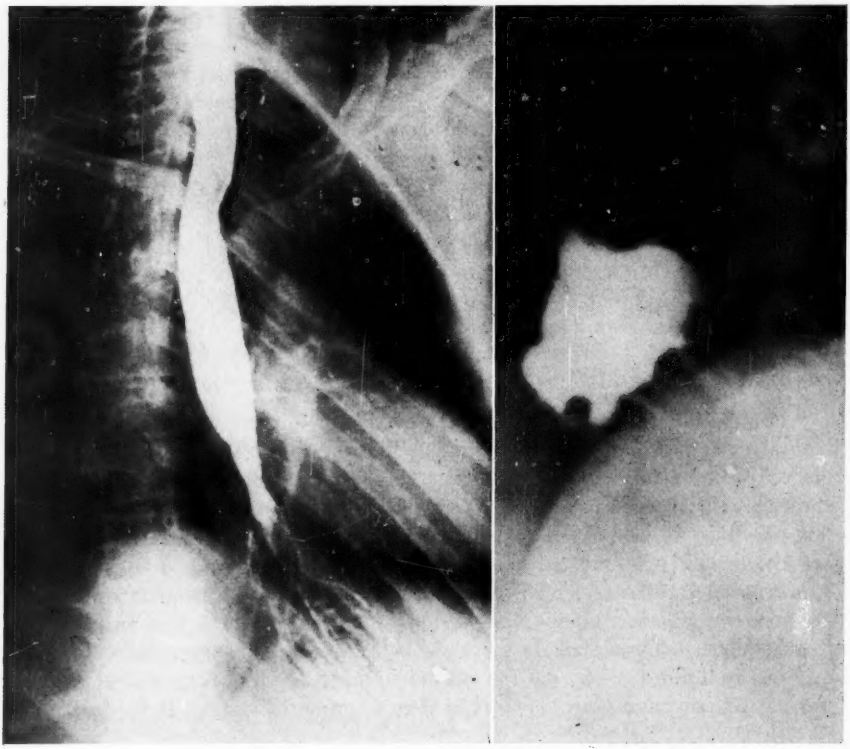

Fig. 1. Fig. 2.

Fig. 1.—Severe esophagitis characterized by an irreducible hiatal hernia with stricture and shortening of the esophagus.

Fig. 2.—Typical x-ray appearance of a concentric, reducible hiatal hernia associated with minimal to moderate esophagitis.

patients with reducible hernias, for the most part, were placed in the moderate or minimal categories (Fig. 2). Patients with erosions or ulcerations determined by esophagoscopy were not necessarily placed in the severe category unless there was evidence of esophageal narrowing or shortening in addition. Esophagoscopy was particularly useful in evaluating patients for placement in either the moderate or minimal categories. Frequently the mucous membrane changes seen in minimal esophagitis were minimal indeed. However, if they were associated with the visible regurgitation of gastric juice on esophagoscopy,

or with some peristaltic abnormalities of the terminal esophagus on x-ray examination, the patients were retained in the study. Since it has been noted that severe pain may exist in the presence of little demonstrable esophagitis, pain alone was not used as a guide in grading the degree of esophagitis present. As a matter of fact, many patients with severe pain, but without good objective evidence of esophagitis, were eliminated from the study. There were likewise discrepancies noted in the size of the hernia, the severity of symptoms present, and the degree of esophagitis demonstrated by esophagoscopy.

Virtually all the cases of esophagitis were of a more or less chronic nature, but admittedly a few were fairly acute, brought on by vomiting, or by dietary or alcoholic excess. All the patients undergoing surgery had esophagitis of a chronic nature in terms of duration of symptoms, and most had been on a previous medical regimen.

PRESENTATION OF MATERIAL

The present report represents the continuation of a study of 55 patients with esophagitis reported in 1956.[8] From January, 1949, to Dec. 31, 1958, 130 patients, ranging in age from 27 to 84 years, have been seen with varying degrees of esophagitis. In 103 (79 per cent) of these patients, a hiatal hernia was present, and in 27 (21 per cent) patients no hiatal hernia could be demonstrated. This distribution is in accord with the reports of other investigators.[6, 7]

The symptoms in these individuals existed for varying periods of time, and were the usual ones of substernal pain radiating through to the back, and heart burn and dyspepsia, usually made worse by food and alcohol indiscretion as well as by lying down. Bleeding was frequently present, rarely as massive hemorrhage, more frequently occult in nature. Dysphagia was not a constant finding. The esophagograms of all the patients were reviewed to confirm the presence or absence of a hiatal hernia and to attempt to correlate the x-ray findings, symptoms, and the findings reported by esophagoscopy. The majority of patients were subjected to esophagoscopy by us to ascertain the degree of esophagitis present. All of the hiatal hernias were of the concentric type, with the exception of two classified as paraesophageal. Two patients have been lost to adequate follow-up examination, and 2 patients died postoperatively, leaving 126 patients in whom the results of treatment could be evaluated.

Factors in the development of esophagitis other than a hiatal hernia were ascertained, and these are listed in Table II. In 103 patients with esophagitis and hiatal hernia, the hiatal hernia was the only apparent cause for the esophagitis in 83, while 20 patients had other significant lesions as listed. Fourteen patients had evidence of an associated gastric or duodenal ulcer, 1 of whom had had a previous cardioplasty. One patient had had a previous gastric resection for duodenal ulcer, and another a gastroenterostomy. In those individuals with esophagitis and no demonstrable hiatal hernia, no specific etiological factor could be found in 10, while the other 17 patients had a specific precipitating event or lesion associated with the development of the esophagitis (Table

TABLE II. ASSOCIATED LESIONS IN ESOPHAGITIS WITH HIATAL HERNIA

No associated lesion	83
Duodenal or gastric ulcer	9
Duodenal ulcer, vomiting	1
Duodenal ulcer, vomiting, gastric tube	2
Duodenal ulcer, cardioplasty	1
Gastric tube	1
Scleroderma	1
Cholecystitis and cholelithiasis	2
Varices, cirrhosis, duodenal ulcer	1
Gastric resection for duodenal ulcer	1
Previous gastroenterostomy	1
Total	103

III). Seven of these patients had evidence of a gastric or duodenal ulcer. Again, 1 patient in this group had had a previous gastric resection for a gastric ulcer.

TABLE III. ASSOCIATED LESIONS IN ESOPHAGITIS WITHOUT HIATAL HERNIA

No associated lesions	19
Gastric or duodenal ulcer	5
Vomiting	
Acute perforation	2
Acute ulcers with bleeding	2
Chronic esophagitis	1
Ulcer, vomiting, gastric tube	1
Cardioplasty	3
Cardioplasty and duodenal ulcer	1
Ectopic gastric mucosa	1
Previous gastrectomy for gastric ulcer	1
Total	27

TREATMENT

The treatment of peptic esophagitis may be medical or surgical. Surgical treatment is reserved for those patients developing the complications of intractable pain, significant bleeding, stenosis, and perforation. Medical treatment has been considered as definite therapy in those patients falling into the following categories: (1) acute esophagitis, such as produced by prolonged and severe vomiting, (2) certain individuals in whom the esophagitis is not associated with a hiatal hernia, and (3) esophagitis in those patients with hiatal hernia in whom elective surgery is contraindicated or refused.

Proper medical therapy has been well established and consists of a bland diet, antacids, elevation of the head of the bed and, if stenosis exists, esophageal dilations with a Jackson-Plummer dilator over a previously swallowed string.

The majority of patients with hiatal hernia received an adequate trial of medical therapy prior to surgery. Hiatal hernia repair was done essentially by the technique described by Allison[1] utilizing the transthoracic approach. In certain instances of short esophagus, transplantation of the esophageal hiatus to the dome of the diaphragm, with associated phrenic crush and extensive freeing of the proximal esophagus for added length, was necessary. The acute angle of entry of the esophagus into the stomach was assured in some of the

patients by suturing the terminal esophagus to the adjacent gastric fundus, while, in others, the fundus of the stomach was simply sutured to the dome of the diaphragm. In no instances were associated drainage operations on the pylorus, gastric resections, or other ancillary procedures performed. Postoperatively, the patients were advanced rather rapidly to a regular diet, maintaining an ulcer regimen only if the symptoms of esophagitis persisted for that short interval of time during which the esophageal lesions were healing. Occasional postoperative dilations were necessary in those individuals with esophageal stricture.

RESULTS

The results of treatment in the 130 patients constituting the basis of this report are summarized in Tables IV and V. Only 2 patients in the entire series were lost to follow-up study. There were 2 postoperative deaths, 1 following closure of a spontaneous perforation of the esophagus, the other following the repair of a hiatal hernia. Morbidity was that usually associated with

TABLE IV. TREATMENT OF ESOPHAGITIS WITH HIATAL HERNIA

RESULTS	DEGREE	CONSERVATIVE TREATMENT	REPAIR HIATAL HERNIA	ESOPHAGO-GASTRECTOMY
Good	Severe	2	25	
	Moderate	1	14	
	Minimal	5	19	
	All	8	58	
Fair	Severe	7	1	
	Moderate	3	1	
	Minimal	4	1	
	All	14	3	
Poor	Severe	6	3	2
	Moderate	2	1	
	Minimal	5		
	All	13	4	2

One patient lost to follow-up.

TABLE V. TREATMENT OF ESOPHAGITIS WITHOUT HIATAL HERNIA

RESULTS	DEGREE	CONSERVATIVE TREATMENT	RECONSTRUCT CARDIAC SPHINCTER MECHANISM	ESOPHAGO-GASTRECTOMY	REPAIR PERFORATION	JEJUNAL TRANSPOSITION
Good	Severe	4		1	1	1
	Moderate	2	5			
	Minimal	3	2			
	All	9	7	1	1	1
Fair	Severe					
	Moderate					
	Minimal					
	All					
Poor	Severe	2		3	1	
	Moderate					
	Minimal	1				
	All	3		3	1	

One patient lost to follow-up.

thoracic procedures. Peculiar to the operation of hiatal hernia repair in this series was the development of temporary dysphagia in a few patients postoperatively when none existed preoperatively. The dysphagia subsided spontaneously, and only occasionally was a single dilatation necessary.

Of the 103 patients with a demonstrable hiatal hernia, 65 had hiatal hernia repair as definitive treatment for the associated esophagitis. The esophagitis in 29 of these patients was graded as severe, and 23 of these had a strictured or short esophagus. Sixteen patients had moderate esophagitis, and 20 had minimal esophagitis. The majority of the patients had significant symptoms, however, regardless of the demonstrable severity of the esophagitis. The results of

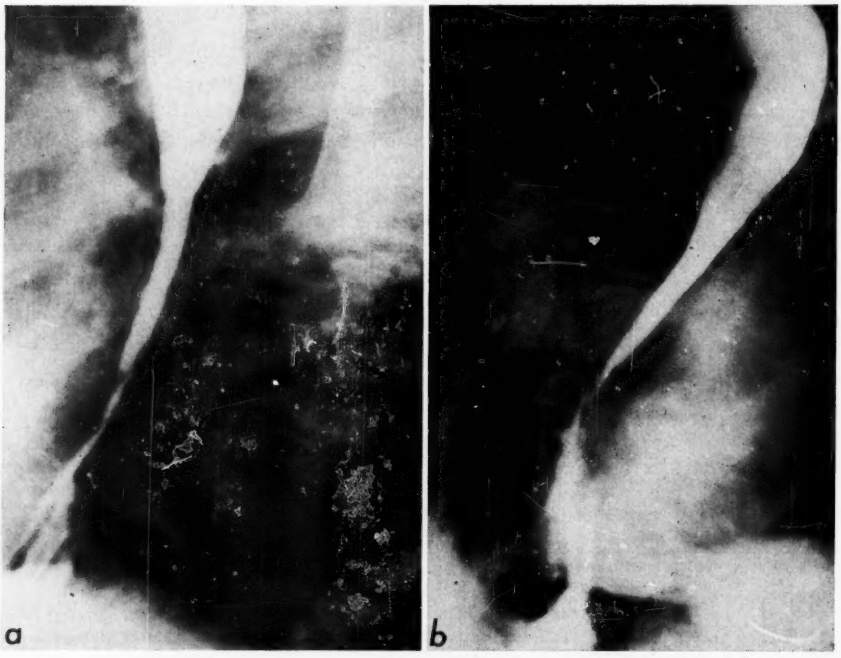

Fig. 3.—*a*, Esophagrams before treatment for esophagitis with stricture and hiatal hernia. *b*, After 2 years of medical therapy during which time thirty-seven esophageal dilatations were done. Although the patient is relatively asymptomatic, there is little change in the x-ray appearance.

surgery in terms of alleviation of prior symptoms, ability to eat all types of food, and absence of the need of special medication or treatment were good in 58 of the 65 patients with hiatal hernia repair. The results were fair in 3 (improvement, but not complete amelioration of symptoms), and poor in 4 (no change in symptoms in 3, and death in 1). The poor results occurred in 3 patients with severe esophagitis, and 1 with moderate esophagitis. Twenty-five of 29 patients with severe esophagitis were completely cured with the ability to eat types of food the ingestion of which for years previously resulted in intolerable symptoms. Patients with moderate or minimal esophagitis

all had good or fair results with the exception of 1. It is interesting to note by way of contrast that in the 35 patients having esophagitis and an associated hiatal hernia treated medically, the results of treatment were good in only 8, fair in 14, and poor in 13. There were 15 patients with severe esophagitis in this group; 13 had fair or poor results, and only 2 had good results. The roentgenograms of one of the patients with a good result are shown in Fig. 3. This patient received 37 esophageal dilatations over a period of 2 years, has only slight dysphagia, and is receiving dilatations about every 6 months at present.

Two patients had esophagogastrectomy performed as the treatment of choice for esophagitis, early in our experience, with poor results. This operation would not be done at the present time.

Twenty-seven patients in whom no hiatal hernia could be demonstrated were treated for esophagitis (Table V). One patient was lost to follow-up, and 1 patient died following repair of a spontaneous esophageal perforation. Twelve patients were treated by the conservative means outlined previously, with good results in 9 and poor results in only 3. Four of 6 patients with severe esophagitis reported good results. Seven patients who had reconstitution of the cardiac sphincter mechanism as the treatment for their esophagitis will be discussed later. Four patients had an esophagogastrectomy, with a good result in only 1 and poor results in the other 3. One patient survived repair of a spontaneous perforation of the esophagus, and 1 patient died following this procedure.

In only one instance so far has a jejunal interposition procedure been done; this in a patient with long-standing esophagitis following a Heineke-Mikulicz cardioplasty for achalasia of the esophagus. The result has been outstandingly good. In the light of this experience, 2 other patients are being considered for this operation at the present time, one having had a previous esophagogastrectomy, and the other a Heineke-Mikulicz cardioplasty.

RECONSTRUCTION OF THE CARDIAC SPHINCTER MECHANISM IN PATIENTS WITH ESOPHAGITIS AND NO DEMONSTRABLE HIATAL HERNIA

Winkelstein,[25] in 1935, described peptic esophagitis as a new clinical entity, stating that the mechanism was not entirely clear. Since that time, a number of articles associating peptic esophagitis with herniation of the stomach through the esophageal hiatus have been published. In 1957, Winkelstein[26] discussed peptic esophagitis associated with duodenal ulcer and differentiated it from that associated with a hiatal hernia, severe vomiting, esophageal intubation, previous operations on the esophagogastric junction, and that associated with persistent embryonic columnar epithelium. He reported 16 patients, 5 of whom had a small associated hiatal hernia. These, he believed, were secondary to traction of the stomach into the chest by a chronically inflamed esophagus. Such an esophagitis might be described as a "primary" or "true" peptic esophagitis, a part of an ulcer diathesis.

Whether an ulcer diathesis exists or not, there must be some explanation for the transcardial reflux of gastric juice. The most logical concept is incompetency of the cardiac sphincter mechanism which exists most frequently

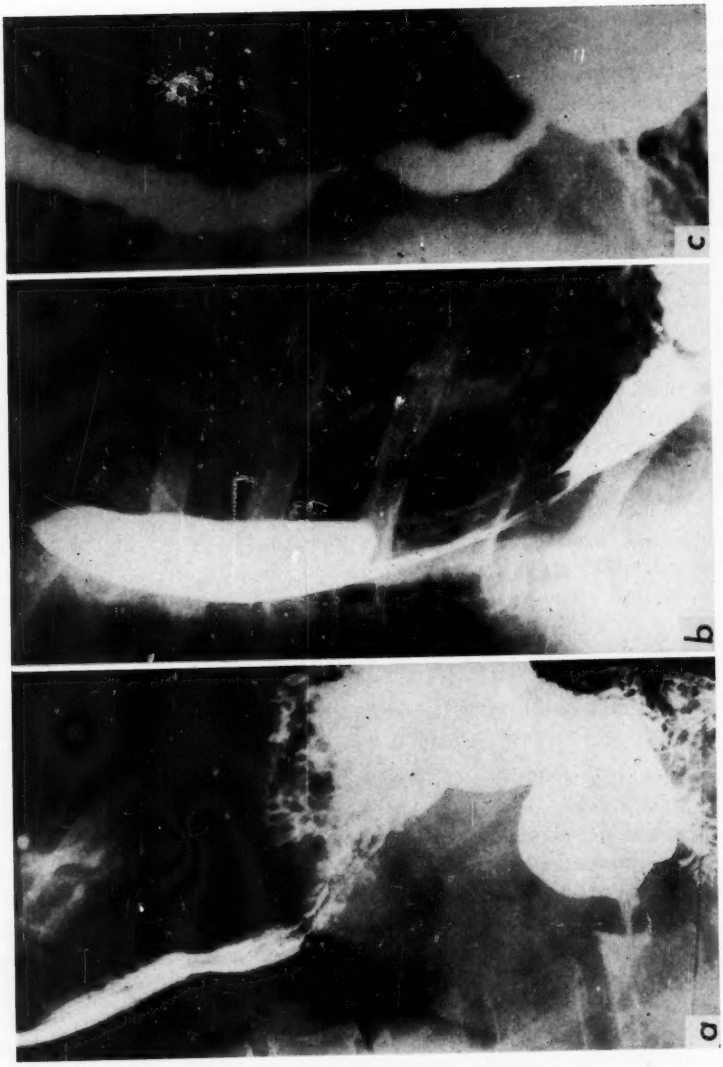

Fig. 4.—Roentgenograms of patients with esophagitis and no demonstrable hiatal hernia in whom reconstitution of the cardiac sphincter mechanism was done.

in association with a hiatal hernia. It is well known that herniation of the stomach through the esophageal hiatus is frequently missed on routine upper gastrointestinal studies, and may be difficult to demonstrate even when special efforts are made. In other instances, although no hiatal hernia is seen, it is possible to demonstrate regurgitation of barium from the stomach into the esophagus, indicating insufficiency of the cardiac sphincter mechanism. There are at least 4 patients in the present series in whom a hiatal hernia was found only after repeated x-ray examinations. Furthermore, we have been intrigued on occasion by the fact that what has been considered a hiatal hernia by us has been interpreted and reported by the radiologist as a phrenic ampulla. This discussion points to the conclusion that small undiscovered hiatal hernias, or at least an anatomic insufficiency of the cardiac sphincter mechanism, are responsible for many cases of so-called "true" peptic esophagitis; and that if a small "traction" hiatal hernia is present, it is likely the cause rather than the result of the esophagitis. It should logically follow that certain patients with esophagitis in whom no hiatal hernia is demonstrated should benefit by reconstruction of the cardiac sphincter mechanism as in hiatal hernia repair. Seven patients in the present study falling into this category of esophagitis with no demonstrable hiatal hernia were operated upon, and the cardiac sphincter mechanism was reconstituted in the usual manner. The results of this procedure in all 7 patients were uniformly good. Three case reports follow of patients who are typical of the group.

CASE 1.—M. B., a 37-year-old white male laboratory worker, had had epigastric and substernal pain 14 years prior to admission. The burning pain was aggravated by drinking alcoholic beverages, dietary indiscretion, smoking, and lying down after meals. The pain was partially controlled for short intervals by diet and antacids. X-ray studies failed to show evidence of the presence of an esophageal hiatal hernia (Fig. 4, *a*). The physical examination was not remarkable. On Dec. 12, 1958, esophagoscopy revealed a minimal chronic esophagitis with free regurgitation of gastric juice into the terminal esophagus. Following this examination, reconstitution of the cardiac sphincter mechanism was done through a transthoracic approach. At the time of surgery the esophageal hiatus was patulous, but no evidence of a hiatal hernia was seen. The postoperative course was uneventful, and the patient was discharged on the ninth postoperative day. He returned to work 2 weeks later and has been entirely asymptomatic to the present time on an unlimited diet containing foods that could not be ingested previously.

CASE 2.—E. D., a 35-year-old white male steel worker, complained of substernal burning of 4 years' duration, aggravated by lying down after meals and at night. He had been on ulcer therapy with little relief for the first 2 years of his illness, and discontinued this therapy when he realized it was not helping. Upper gastrointestinal studies in another hospital showed no evidence of a hiatal hernia, gastric, or duodenal ulcer. On two prior hospital admissions, esophagoscopy disclosed esophageal erosions in the terminal esophagus. He entered the hospital for operation complaining of an increase in his symptoms for the previous 3 months. Physical examination was noncontributory. X-ray studies again failed to demonstrate a hiatal hernia (Fig. 4, *b*). Esophagoscopy disclosed minimal esophagitis with regurgitation of gastric juice. At operation on April 22, 1957, it was noted that the esophageal hiatus was patulous. There was a minimal bulging of the stomach through the diaphragm. His postoperative course was uneventful except for a hydrothorax on the left which cleared with a single aspiration. He was discharged from the hospital on the thirteenth postoperative day. He has been asymptomatic to the present time except for some residual chest wall pain.

CASE 3.—A. H., a 35-year-old white male salesman, was admitted to the hospital with a 9-month history of substernal distress and postprandial pain, particularly aggravated by alcoholic intake which was seemingly a necessary part of his job. Physical examination was noncontributory. At esophagoscopy on March 24, 1956, a moderate esophagitis with linear erosions was noted. X-ray studies at this time showed no evidence of an associated hiatal hernia (Fig. 4, *c*). He was placed on appropriate medical therapy with no satisfactory relief and he returned for operation on May 28, 1956. At operation the esophageal hiatus was lax, and a very small but definite hernia was seen. The postoperative course was satisfactory, and he was discharged from the hospital on the thirteenth postoperative day with complete relief of his symptoms. He has returned to his prior occupation as a salesman with no recurrence of symptoms to date.

Definite esophagitis of a minimal or moderate degree associated with visible reflux of gastric juice was confirmed in all these patients by esophagoscopy. At the time of operation, definite anatomic changes were noted in the region of the esophageal hiatus in all 7. These consisted primarily of relaxation of the muscles constituting the right crus of the diaphragm making the esophageal hiatus more patulous than normal. A small portion of stomach protruded through the hiatus into the chest in 4 patients. No visible evidence of herniation was present in the other 3. In spite of such minimal anatomic findings, all of these patients benefited from surgical reconstruction of the cardiac sphincter mechanism with complete alleviation of the preoperative symptoms.

DISCUSSION

An attempt has been made to present in specific terms what is thought to be the proper treatment of peptic esophagitis. Obviously, all therapy must have as its end the inhibition of the damaging effects of acid-peptic juice on the vulnerable esophageal mucosa. To accomplish this, such contributing factors as an associated duodenal ulcer, the presence of a hiatal hernia, or previous surgery at the esophagogastric junction must be assessed in the light of their specific contribution to the production of the esophagitis. It must be decided whether the esophagitis can be improved by simple neutralization of the regurgitant acid juices as with the usual medical therapy, whether the acid-peptic juice itself must be eliminated by destroying the secretory potential of the stomach as in extensive gastric resection, or, finally, whether correction of an abetting factor such as an associated hiatal hernia will be sufficient to prevent further esophagitis. If the esophagitis can be traced to a single initiating event such as an episode of severe vomiting, or the presence of a gastric tube during the postoperative period, in which the insult to the esophagus is not likely to be a continuous or repetitive one, conservative therapy may well be considered as definitive treatment. In instances of chronic esophagitis with a more insidious onset, every attempt should be made to demonstrate the presence of an associated hiatal hernia or incompetency of the cardiac sphincter mechanism. If the presence of a hernia is demonstrated, medical therapy has been unsatisfactory, in our experience, in that only 8 of 35 patients obtained good results. Contrariwise, patients without an associated hiatal hernia were treated fairly successfully by medical means. It is believed that the poor results in the former group are due to the continued presence of the hiatal hernia as an abetting

factor. In spite of this, patients should always be given a trial of medical therapy prior to operation, not only because a few will respond satisfactorily, but also because in most instances there will be some regression of the esophagitis making the contemplated operation easier to accomplish.

In those instances in which the esophagitis is associated with a reducible hernia, it is generally accepted that repair of the hernia is the treatment of choice. However, some difference of opinion exists as to the proper treatment of those patients with a short or strictured esophagus associated with an irreducible hernia. Twenty-nine patients in the present series had severe esophagitis, and 24 of these had a stricture or short esophagus demonstrable by x-ray studies and confirmed by esophagoscopic findings. By employing the type of hernia repair described previously, it was possible to obtain good results in 20 of these patients, with poor results in only 3. One of the failures occurred in a patient whose symptoms were aggravated by a large alcoholic intake. In another, a previous gastrectomy for duodenal ulcer indicated the possibility of a rather intractable situation. In a third, death occurred postoperatively. It is truly remarkable to note the rapid regression of symptoms and signs of esophagitis once the esophagogastric junction is replaced below the diaphragm to break the vicious cycle of acid-peptic irritation of the esophagus. In view of such satisfactory results with a procedure that neither changes the normal physiology nor the functional capacity of the upper gastrointestinal tract, extensive gastric surgery or plastic procedures on the esophagogastric junction do not seem justified as a primary therapeutic effort. There are exceptions to any rule, however. Instances in which the terminal esophagus is destroyed by chronic irritation and inflammation, to the extent that it must be resected, occasionally exist.

There were 14 patients in the present series with evidence of a gastric or duodenal ulcer associated with a hiatal hernia, suggesting either an ulcer diathesis or the possibility of a gastric outflow obstruction as an additional abetting factor. In no instance was more than hiatal hernia repair necessary to obtain a good result in any of these patients, although in one a simple Ramstedt type pyloroplasty was contemplated.

Probably the most serious problems in treating esophagitis arise in those individuals who have a severe esophagitis resulting from previous surgery on the esophagogastric junction. Our attempts at treating esophagitis by esophagogastrectomy resulted in almost uniform failure, with continuation of the esophagitis. Our most successful experience with this problem recently has been with 1 patient in whom a jejunal interposition procedure was done with a result gratifying enough to encourage contemplation of the continued use of the procedure in 2 more patients in the near future. We favor this operation to that proposed by Ellis,[12] since the latter procedure leaves gastric mucosa in juxtoposition to that of the esophagus, which past experience has shown creates the potential for further esophagitis.[10, 15]

The technique of hiatal hernia repair to reconstitute the cardiac sphincter mechanism in certain individuals with intractable reflux esophagitis, in whom

no hiatal hernia can be demonstrated, has been used with gratifying results. This may seem like a radical approach to the problem at first glance. However, if one concedes that peptic esophagitis is the result of regurgitation of acid-peptic juices from the stomach, accepts the well-known fact that a small hiatal hernia or reflux may be difficult to demonstrate roentgenologically, and accepts the satisfactory results obtained by the reconstitution of the cardiac sphincter mechanism as in hiatal hernia repair, then this approach to the treatment of peptic esophagitis without demonstrable hernia seems reasonable. The good results in 7 patients treated in this manner, all of whom had been on prolonged medical therapy prior to surgery, strengthens this contention.

SUMMARY

1. One hundred and thirty patients with peptic esophagitis are presented. One hundred and three had a demonstrable hiatal hernia, and in 24 no associated hernia was found.

2. Associated etiological factors, such as severe vomiting, the presence of a duodenal ulcer, or previous operations on the esophagogastric junction, are discussed.

3. Repair of the associated hiatal hernia, rather than one of the other more complicated procedures recommended recently, is suggested as the treatment of choice, even in those patients with stricture and shortening of the esophagus associated with an irreducible hernia.

4. As a new concept in the treatment of esophagitis, reconstruction of the cardiac sphincter mechanism as in hiatal hernia repair is suggested, even though no definite hiatal hernia has been demonstrated, in certain selected patients in whom long-term medical therapy has failed.

REFERENCES

1. Allison, P. R.: Reflux Esophagitis, Sliding Hiatal Hernia, and the Anatomy of Repair, Surg. Gynec. & Obst. **92**: 419, 1951.
2. Allison, P. R., and daSilva, L. T.: The Roux Loop, Brit. J. Surg. **41**: 173, 1953.
3. Arroyave, R., Clatworthy, H. W., Jr., and Wangensteen, O. H.: Experimental Production of Esophagitis and Esophageal Ulcers in Dogs, S. Forum (1950), pp. 57-59, 1951.
4. Aylwin, J. A.: The Physiological Basis of Reflux Oesophagitis in Sliding Hiatal Diaphragmatic Hernia, Thorax **8**: 38, 1953.
5. Barrett, N. R.: Chronic Peptic Ulcer of the Oesophagus and "Oesophagitis," Brit. J. Surg. **38**: 175, 1950.
6. Benedict, E. B., and Nardi, G. L.: Peptic Stenosis of the Esophagus, Am. J. Surg. **93**: 238, 1957.
7. Carver, G. M., Jr., and Sealy, W. C.: Peptic Esophagitis, A. M. A. Arch. Surg. **68**: 286, 1954.
8. Cross, F. S., and Kay, E. B.: The Etiology and Treatment of Peptic Esophagitis, Ann. Surg. **143**: 360, 1956.
9. Cross, F. S., Kay, E. B., and Johnson, G. F.: Neuromuscular Imbalance of the Esophagus Associated With Hiatal Hernia as Studied by Means of Cinefluorography and Intraluminal Pressure Recordings, J. THORACIC SURG. **34**: 736, 1957.
10. Cross, F. S.: Unpublished data.
11. Effler, D. B., and Ballinger, C. S.: Complications and Surgical Treatment of Hiatus Hernia and Short Esophagus, J. THORACIC SURG. **22**: 235, 1951.
12. Ellis, F. H., Jr.: A Physiologic Operation for Ulceration and Stricture of the Terminal Esophagus, Proc. Staff Meet. Mayo Clin. **31**: 615, 1956.

13. Ferguson, D. J., Sanchez-Palomera, E., Sako, Y., Clatworthy, H. W., Jr., Toon, R. W., and Wangensteen, O. H.: Studies on Experimental Esophagitis, Surgery **28**: 1022, 1950.
14. Hale, H. W., Jr., and Drapanas, T.: Reflux Esophagitis, Am. J. Surg. **93**: 228, 1957.
15. Hoag, E. W., Kiriluk, L. B., and Merendino, K. A.: Experiences With Upper Gastrectomy, Its Relationship to Esophagitis With Special Reference to the Esophagogastric Junction and Diaphragm. Study in Dog, Am. J. Surg. **88**: 44, 1954.
16. Jones, J. C.: Discussion of a paper by Hoag et al.[15]
17. MacKenzie, M.: Diseases of the Throat and Nose, London, 1884, J. & A. Churchill.
18. McLean, L. D., and Wangensteen, O. H.: The Surgical Treatment of Esophageal Stricture, Surg. Gynec. & Obst. **103**: 5, 1956.
19. Moersch, R. N., Ellis, F. H., Jr., and McDonald, J. R.: Pathologic Changes Occurring in Severe Reflux Esophagitis, Surg. Gynec. & Obst. **108**: 476, 1959.
20. Ochsner, A.: Discussion of a paper by Effler and Ballinger.[11]
21. Quinke, H.: Quoted by Moersch et al.[19]
22. Skinner, H. H., and Merendino, K. A.: An Experimental Evaluation of an Interposed Jejunal Segment Between the Esophagus and the Stomach Combined With Upper Gastrectomy in the Prevention of Esophagitis and Jejunitis, Ann. Surg. **141**: 201, 1955.
23. Stewart, J. D., Chardack, W. M., and Alfano, G. S.: Reflux Esophagitis, Ann. Surg. **141**: 627, 1955.
24. Sweet, R. H., Robbins, L. L., Gephart, J., and Wilkins, E. W., Jr.: The Surgical Treatment of Peptic Ulceration and Stricture of the Lower Esophagus, Ann. Surg. **139**: 259, 1954.
25. Winkelstein, A.: Peptic Esophagitis: New Clinical Entity, J. A. M. A. **104**: 906, 1935.
26. Winkelstein, A.: Peptic Esophagitis With Duodenal Ulcer, Am. J. Surg. **93**: 234, 1957.

Discussion

DR. BEATTY H. RAMSAY, Santa Monica, Calif.—I would like to compliment Dr. Cross on his paper, with which I take no exception.

Recently, in reading a book about the Nautilus submarine, Dr. Rickover was said to have interviewed officers and put them into precarious situations unexpectedly and asked them what they would do about it. One such time he told an officer that he had just been put on a lifeboat with 4 other men, only one of whom could live. How could he talk the other 4 into allowing himself to be the survivor?

I was recently brought into a situation I had not experienced before with regard to esophagitis. A 46-year-old patient had never sought medical care, although for 4 years he had had severe obstruction on swallowing. At the time he was seen he was unable to swallow at all and had lost 20 pounds of weight.

The barium study showed a marked constriction of the lower esophagus 4 inches above the cardia, and through this very little barium could pass. An adequate study for hiatus hernia could not be made. Esophagoscopy was perfectly normal as far as his obstructing lesion, but at this point there was a very rigid stenosis and dilatation was impossible. A No. 20 bougie tip could be passed through into the stomach, but dilatation did not occur, and the bougie tip was tightly grasped. There was no sign of esophagitis above this lesion at all.

We thought it could be malignant, but the 4-year history, of course, suggested an inflammatory etiology. An attempt was made to treat him by conservative methods so that a better study could be obtained. He had never had any form of antacid therapy.

However, in the hospital, with all the help we could give him, he was unable to swallow even his saliva. We were therefore in the position of having to do something about his dysphagia.

At surgery we did a linear esophagomyotomy in the Heller fashion, in order to determine the pathology. We obtained a biopsy of the entire thickness of the fusiform lesion from the external musculature down to and including the mucosa. The outstanding feature here was an eccentric lesion, 2 inches in length, where the pathology appeared to be edema within the muscle coat, with chronic and acute inflammation of the mucosa.

In essence, there was fibrosis of years' duration with acute inflammation and ulceration. No malignancy having been found, the question was, what to do? Should we do an

extensive resection on this man who had never had any therapy before but who could not swallow?

There was an hiatus hernia sac present but nothing was contained therein.

All we did was suture the mucosa very carefully, leaving the muscle apart as in the typical Heller esophagomyotomy.

The esophageal lumen was now large enough to accommodate a No. 18 Levin tube and a finger; therefore it seems that the obstructing factor was in the muscle wall, mostly due to edema and was not mucosal constriction. There was no problem during the postoperative course. Relief of the mural obstructive factor by simple longitudinal myotomy, plus correction of the hiatus hernia or other primary cause, may prove to be effective treatment for inflammatory strictures of the lower esophagus and thus save these people from extensive surgical resection.

DR. MAX G. CARTER, New Haven, Conn.—I should like to congratulate the authors. I think they have done a splendid job, and I don't think they need to feel any temerity at all about suggesting surgery for the patient who has peptic reflux esophagitis, because we all know it is a bad disease.

I merely would like to make a suggestion which many of you know about, I am sure, for refining the methods of diagnosis in that group of patients in which hiatus hernia is not obvious. Most of us now have improved radiologic facilities because of the advances in cardiovascular surgery. Image amplification with cineradiography is very useful for study of the barium swallow. A moderate number of patients in whom the diagnosis of esophageal vestibule or ampulla would otherwise be made will be found to have very small short esophagus-type hiatus hernias. This I am sure is the group in which the authors found great improvement, although their radiologists were unable to demonstrate hernias by the traditional methods.

DR. ALFRED HURWITZ, Brooklyn, N. Y.—I would like to compliment Dr. Cross and his associates for a thought-provoking and lucid paper.

There is a law akin to Parkinson's law which I think can be applied to this problem: namely, that the success rate of any operation varies in inverse proportion to the number of operations that are currently employed. I think, however, that many of the operative procedures that have been devised have a good deal of efficacy, and with that in mind, during the past 2 years I have combined salient features of operations that have been employed in the past.

I hesitate to mention before this Society my preference for the transabdominal approach. Even in the obese patient, I have had little difficulty in exposing the diaphragmatic aperture. I prefer to do the repair in the manner described by Harrington. I find that I can reach the more caudal aspects of the aperture more easily by the transabdominal approach. In addition, exploration of the abdominal organs can be carried out and cholecystectomy can be performed if indicated.

Then I reconstitute the angle, an approach which Mr. Barrett has emphasized, by placing two or three sutures between the medial wall of the fundus and the left lateral wall of the esophagus. The fundus is then anchored to the undersurface of the diaphragm with several additional sutures.

If the patient has an elevated gastric acidity, a bilateral vagectomy is done at this time. A Heineke-Mikulicz pyloroplasty is performed in all instances. This procedure constitutes another reason for my preference for the abdominal approach: The pylorus is sutured in a transverse direction with interrupted silk sutures of the Gambee type, a method employed for many years by Weinberg of Long Beach.

Our series is too small and the follow-up period too short to be statistically significant but our results thus far have been most gratifying.

One final word, and that is that the iatrogenic complication of esophagitis, secondary to Levin tube intubation, can be eliminated by the use of gastrostomy tubes, and we are using them with increasing frequency.

DR. WILLIAM E. ADAMS, Chicago, Ill.—I would like to say just a word about a factor that has already been mentioned, namely the question of malignancy arising in these patients.

We have recently reviewed 32 cases of the short esophagus type, in 8 of which there was a malignancy arising on the stomach side of the esophagogastric junction. I am sorry to add that in those patients that were treated longer than 2 years ago none lived longer than that time.

DR. CROSS (Closing).—I would like to express my appreciation to those individuals who discussed this paper, and I would especially like to re-emphasize what Dr. Carter stated about small hiatal hernias being present when they are not demonstrable by ordinary examination. This is one of the important points of the presentation; that is, that small hernias may be present, although not demonstrated, and that such patients can be helped by hiatal hernia repair. Our colleagues in internal medicine categorize these patients as having "true" or "primary" esophagitis, and treat them medically, frequently without too much success.

The second important point that I would like to re-emphasize is that I think we have shown that even severe esophagitis can be treated successfully by surgical repair of the associated hiatal hernias, and that other ancillary procedures, such as drainage operations, vagotomy, etc., are not necessary, and they may even complicate the result.

UTILITY OF A NEW PROCEDURE OF VALVULAR ESOPHAGOGASTROSTOMY IN CASES OF BRACHYESOPHAGUS AND STRICTURE: CLINICAL AND EXPERIMENTAL STUDIES OF CIRCUMFERENTIAL ESOPHAGOFUNDOPLASTY

David H. Watkins, Ph.D., William R. Rundles, M.D. (by invitation), and Louis Tatom, M.D. (by invitation), Denver, Colo.

REGURGITATION ESOPHAGITIS and peptic ulceration at the esophagogastric junction is an important problem at the present time, despite extensive experimental and clinical investigations to elucidate the mechanisms of development of the lesion and despite ingenious suggestions for operative management.

Since Quincke[1] in 1879 inculpated the gastric digestive juices as being the principal agents producing esophagitis, a continuing, accumulating body of information tends to confirm this opinion. Esophagitis may be produced in rats by the simple expedient of pyloric ligation. Esophagitis has been produced by implanting gastric mucosa into the esophagus, and acute, experimental ulcers of the esophagus have been shown to be retarded in their healing in the presence of hydrochloric acid. The presence of esophagitis has been correlated with the regurgitation of acid peptic secretions.

The physiology of the cardia is still not completely explained. The different experimental investigations and clinical observations, however, suggest that this closing mechanism is dependent on several factors. Reflux is prevented normally in five ways:

1. By the oblique implantation of the esophagus into the lesser curvature of the stomach like the tip of a whistle, where the esophagus forms with the fornix of the stomach the acute angle of His. (The mucosal fold produced is called the valve of Gubaroff.)

2. By the muscle fibers of the stomach along the lesser curvature which lie like a sling surrounding the cardia. This loop of gastric muscle, just deep to the mucosa, which passes around the cardia and down each side of the lesser curvature helps maintain the angle of His at the incisura. The earliest description of this muscle comes from Thomas Willis of Oxford, who not only described the loop but showed it in a drawing dated 1674.

3. By the muscle fibers of the right crus of the diaphragm which stabilize and support the right border of the esophagus.

From the Professorial Surgical Unit and the Division of Surgery, Denver General Hospital.

Read at the Thirty-ninth Annual Meeting of The American Association for Thoracic Surgery at Los Angeles, Calif., April 21-23, 1959.

4. By the fact of a tonic action of the esophagus or of a sphincter muscle in the region of the cardia, which is found in a state of permanent tonus. This has been a point of continuing argument between anatomists and physiologists, but in my opinion there is no clear-cut evidence of such a sphincter.

5. By another structure which exists in man to oppose the tendency for the cardia to pass into the posterior mediastinum when the diaphragm is relaxed in expiration. This is the diaphragmatico-esophageal membrane or elastic ligament, a modification of the abdominal aponeurosis of the diaphragm (and therefore relatively fixed at its origin!); it bridges the esophageal hiatus and is inserted into the adventitia of the gullet as a membranous ring with upward and downward "cuff" extensions.

Or, using another formula, these different groups of muscles and fascias serve essentially two functions: to preserve or to exaggerate the acute angle of entrance of the esophagus into the stomach, and to close the lower end of the esophagus like a sphincter. We have therefore a kind of valve action and a kind of sphincter action.

The incompetence of the cardia and esophagitis associated with esophageal hiatal hernia are automatically corrected when the now fairly standardized methods for replacing the stomach below the diaphragm and correcting the hiatal defect are applied. What are these principles to be applied to the surgical repair of an ordinary sliding esophageal hiatal hernia in which the esophagus has its normal length (as determined by esophagoscopy) and in which the stomach can be easily replaced within the abdominal cavity? (1) Reduce the hernia to prevent reflux. (2) Diminish the size of the esophageal hiatus to prevent recurrence of the hernia. (3) Reconstitute the esophagogastric angle by fixing the cardia below the diaphragm and by diaphragmatico-fundopexy to allow the fundus to balloon up under the dome of the diaphragm.

In cases of esophageal hiatal hernia in which peptic ulcer has progressed to stricture formation and where the stricture is tough, fibrous, and incapable of dilatation to full caliber, more complicated therapy is indicated. In these cases, the cardia and affected esophagus must be resected and the digestive continuity re-established. The operation of Wangensteen may be performed by resecting the entire acid-bearing area of the stomach, and creating an esophago-antral anastomosis with an extramucosal pyloromyotomy. The difficulty with this operation is the loss of the gastric reservoir. The operation of Allison may be recommended in certain cases. Here, following a subtotal gastrectomy which leaves only the gastric antrum, continuity is re-established by an esophagojejunostomy with the Roux-en-Y. The criticism of this procedure is the absence of the gastric reservoir, and short-circuiting of the duodeno-pancreaticobiliary block. Recent studies have suggested that the interposition of an isoperistaltic segment of jejunum, ileum, or colon between the esophagus and stomach may prevent regurgitation of gastric or duodenal contents into the esophagus and hence avoid esophagitis.

While these methods are all extremely useful in given situations, it would appear that a technique which would allow resections of strictures of the lower third of the esophagus together with preservation of the stomach by a simple

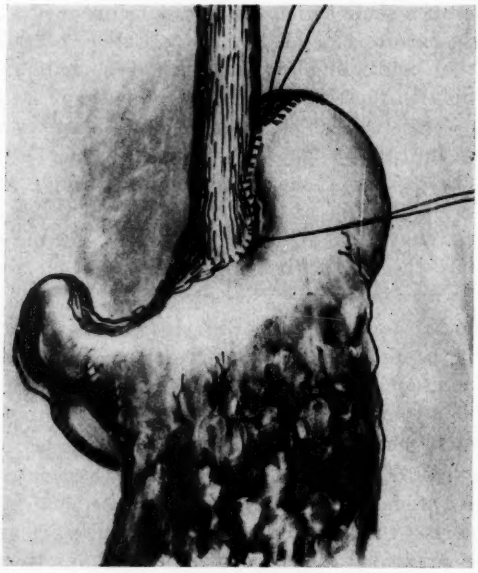

Fig. 1.—The esophagogastrostomy has been completed, and the left border of the esophagus is being approximated to the lesser curvature of the fundus superior to the esophagogastrostomy.

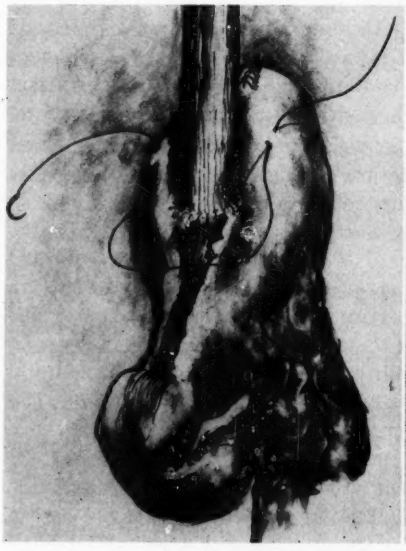

Fig. 2.

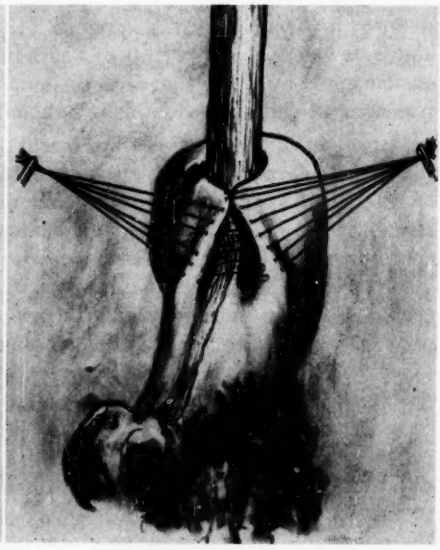

Fig. 3.

Fig. 2.—The first step in performing circumferential esophagofundopexy consists of creating a Witzel type tunnel for the esophagus by approximating the anterior and posterior walls of the stomach with interrupted sutures.

Fig. 3.—The sutures placed to form the circumferential esophagofundopexy are being tied.

surgical technique would be much preferable because these lesions are frequently found in old, debilitated, starved people. Thus, our attention was drawn to investigation of a simple method by which esophagogastrostomy would be performed in such a fashion that reflux esophagitis would be prevented.

MATERIALS AND METHODS

I. Fifteen adult mongrel dogs were prepared. The abdomen was opened through a left rectus muscle retracting incision, and the fundus of the stomach was resected, making certain that the sling muscle passing around the esophagogastric junction at the angle was completely interrupted. The stomach wall was sutured, utilizing two layers of sutures.

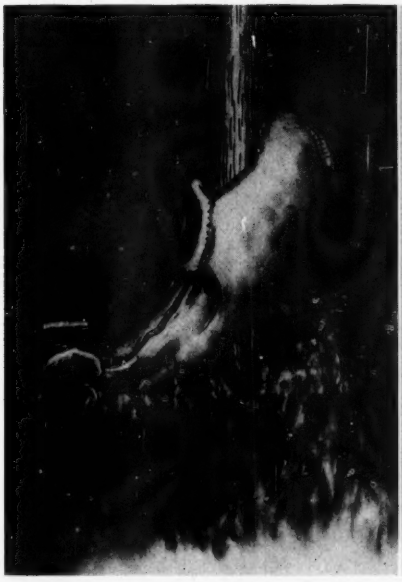

Fig. 4.

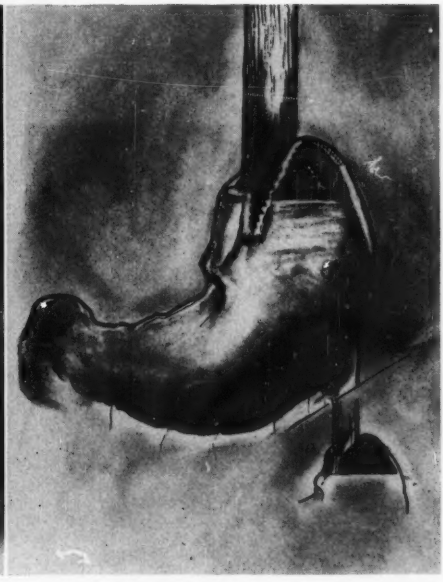

Fig. 5.

Fig. 4.—The completed appearance of the esophagofundopexy is well shown. The approximation of the anterior and posterior walls of the stomach must extend well below the junction of the esophagus and stomach.

Fig. 5.—Frontal section showing the disposition of the esophagus with relation to the wall of the stomach and the magenblase. The insert shows the approximation of the esophageal walls at the position of rest (absence of swallowing).

II. Ten dogs were prepared for controls by placing the cardioesophageal junction above the diaphragm by interrupting the diaphragmatico-esophageal membranes, and thus creating a sliding type of esophageal hiatal hernia. These animals were observed over a period of 18 months. The edges of the hiatus were sown to the prolapsed stomach with interrupted silk sutures. In a few cases the hiatus required enlargement by an incision through the hiatal ring.

III. Twenty dogs were prepared as follows. Through a left transthoracic incision the esophagus was identified, the esophageal hiatal ring was incised in a radial direction for 2 to 3 cm., and the stomach was drawn up into the left pleural cavity. The cardioesophageal angle was reconstructed above the diaphragm by approximating the medial border of the gastric fundus to the left border of the esophagus with a row of interrupted sutures of 3-0 silk for a distance of 5 to 10 cm. from the cardioesophageal junction (Fig. 1). With the angle thus reconstructed, circumferential esophagofundopexy was carried out. The anterior and posterior surfaces of the fundus are wrapped around the distal esophagus for a distance of 4 to 6 cm. and approximated by interrupted silk 3-0 sutures to produce in essence a Witzel tunnel (Figs. 2, 3, 4, 5).

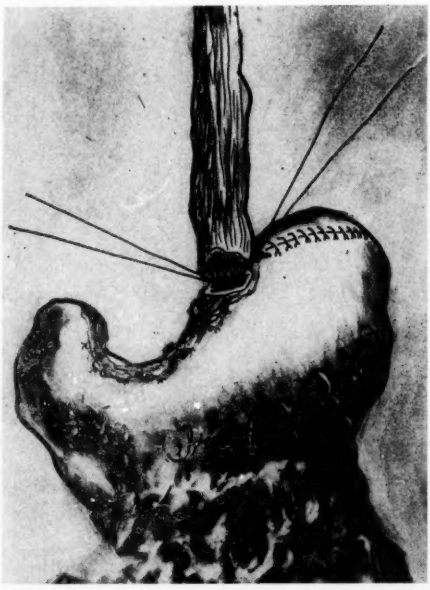

Fig. 6.—A stricture involving the lower third of the esophagus has been excised, and the inosculation of the esophagus into the cardia has been obliterated by suture. An esophagogastrostomy is being performed on the lesser curvature.

Thus, the esophagogastric junction, angle, and Witzel tunnels were left in the left pleural cavity with the diaphragm loosely sutured to the stomach below. The diet was gradually increased from fluids to full kennel ration.

In 10 dogs the same plan of procedure was carried out except that the distal 4 cm. of esophagus were resected and esophagogastrostomy was carried out (Fig. 6) utilizing the same plan of circumferential esophagofundopexy. Dogs with these esophagogastric anastomoses received nothing by mouth for the first 3 postoperative days.

Esophagoscopy was routinely performed under Pentothal anesthesia preoperatively, 4 to 6 weeks postoperatively, and at intervals thereafter until the animal was sacrificed. After the first esophagoscopy, a nasogastric tube was

placed into the stomach and 200 c.c. of a thin barium suspension was injected. Reflux was determined by fluoroscopy, utilizing a tilt-table in the Trendelenburg position and manual pressure on the abdomen.

IV. Fifteen normal fresh postmortem human esophagi and stomachs were studied. The pylorus was cannulated and the stomach was distended with water from a reservoir which was raised alongside a measured rule until the water was seen to escape from the esophagus. These experiments were carried out with the fundus of the stomach in different positions in the body and following removal from the body. In six stomachs a sham operation was performed obliterating the angle of His initially and then carrying out circumferential esophagofundopexy (Fig. 7). Studies were made observing regurgitation following cannulation of the pylorus, and roentgenographic studies were carried out utilizing thin suspensions of barium and distention of the stomachs with air.

Fig. 7.—Appearance of esophagofundopexy performed on the esophagus and stomach removed from a cadaver.

RESULTS

In the group of animals in which fundusectomy was carried out, and in which the loop of Willis remained intact, no esophagitis developed over a period of 12 months. On the other hand, in those animals in which the loop of Willis was interrupted during the fundusectomy, esophagitis appeared promptly, usually within 4 to 6 weeks.

Peptic esophagitis does not develop so regularly following the production of an experimental hiatal hernia by drawing two thirds of the stomach into the thorax and fixing it to the cut edges of the diaphragm by suture as it does

following esophagogastrostomy or by surgical excision of the left crus of the canine diaphragm (which is comparable to the right crus of the human diaphragm) and allowing a cardiac incompetence to develop gradually. The development of esophagitis may be hastened by obstruction of the superior gastric loculus. In any case, however, evidence of peptic esophagitis developed within 9 months in three quarters of these animal preparations.

The series of dogs which had been prepared by circumferential esophagofundopexy showed no evidence of peptic esophagitis when they were observed over a period of 36 months. During esophagoscopy in these animals slight to moderate resistance was encountered in the distal esophagus. With slight pressure the esophagoscope passed through this area, and its passage was sometimes followed by escape of gas or fluid. All of these animals demonstrated competency with the barium studies, whereas the hiatal hernias all exhibited reflux. However, very little pressure was required to inject the thin barium contrast medium through the esophagi into the stomachs of anesthetized dogs which had had this operative procedure.

Our pressure injection experiments confirmed the importance of the mechanical arrangement of the fundus which, when it is excluded from the lumen of that viscus, diminishes greatly the resistance offered by the cardia to regurgitation. When the left leaf of the diaphragm was removed to allow the fundus to protrude into the left pleural cavity unhindered, the pressure necessary to induce regurgitation was increased. The liver tunnel, the crura of the diaphragm, and the pressure of the surrounding viscera are important in maintaining the relationship of the fundus to the body of the stomach and the esophagus and the efficiency of the cardioesophageal angle.

The procedure of approximating the right border of the fundus of the stomach to the left border of the esophagus by a row of interrupted sutures increases the resistance of the cardia to regurgitation only if the stomach and esophagus remain in their relative normal positions within the abdomen. If this same preparation is carried out on the isolated esophagus and stomach, pressure applied through the pylorus produces bulging of the right wall of the esophagus and offers no hindrance to regurgitation. In the normal body, however, the crura, the liver tunnel, and other surrounding structures buttress the right border of the esophagus so that the angular mechanism is efficient.

Pressure regurgitation studies carried out on the isolated esophagus and stomach which had been subjected to circumferential esophagofundopexy showed maintenance of competency even though the entire preparation had been removed from the body (Fig. 8). This is illustrated in Fig. 9 in which the stomach and esophagus have been coated with a thin barium suspension and air has been introduced through the pylorus. The competency of the cardiac valve is seen.

CLINICAL APPLICATIONS

Since the primary pathologic feature in reflux esophagitis is an incompetent cardiac sphincter, the development of esophagitis with its several complications is a secondary phenomenon consequent to sphincter relaxation. Causal therapy

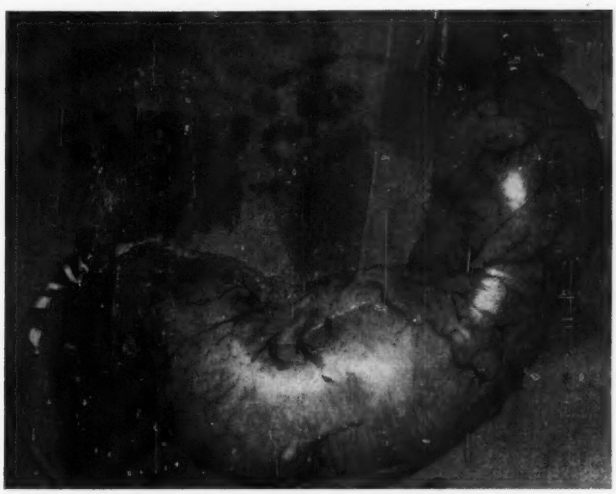

Fig. 8.—The stomach upon which an esophagofundopexy has been performed has been grossly distended with air injected through a catheter passed through the pylorus. Note the competence of the circumferential esophagofundopexy-type valve which allows no air to pass from the stomach into the esophagus.

Fig. 9.—Note the position of the lumen of the esophagus, through which dense barium suspension is passing from the esophagus into the stomach. The area of the esophagofundopexy appears rather coated, but the barium in the lumen is clearly seen.

therefore must be directed to the sphincter mechanism itself. Regurgitation and belching are common physiologic events in normal men and dogs, and they demonstrate that the cardiac mechanism is a reversible one which may be at times incompetent normally. Thus, the simple reflux of gastric chyme is no indication for therapy. This reflux of gastric content may occur with and without esophageal hiatal hernia, and with or without the serious complications of esophagitis. Thus, the procedure would certainly not be considered in patients with reflux esophagitis associated with sliding hiatus hernia which is readily curable by conventional methods of hiatal repair. The procedure which we are considering is suggested only in patients with the serious complications of esophagitis, such as stricture and/or ulceration, hemorrhage, and perforation. Esophagitis occurs frequently in patients with congenitally short esophagus.

Reflux esophagitis is not a single pathologic entity but a group of conditions of probable common origin. Despite the severity of the disease processes in some cases, the inflammatory process may be remarkably superficial. It is common for inflammation and scarring to penetrate the submucosa, even when a marked stricture is present. In these cases rapid epithelial healing follows reduction of the acid gastric secretions which are regurgitated into the esophagus. For the small number of patients who have extension of the inflammatory process into the muscular tunic, the prognosis is less favorable.

In cases of sliding esophageal hiatal hernia with secondary short esophagus, two situations present themselves. In the first case, the esophagus can be dilated readily to good and sufficient caliber. Here we believe transthoracic circumferential esophagofundopexy with no attempt to reduce the stomach below the diaphragm should be carried out. Occasional esophageal dilatations may be required. In cases of esophageal hiatal hernia in which the peptic ulcer has progressed to stricture formation and in which the stricture is tough, fibrous, and incapable of dilatation to full caliber, we believe that distal esophagectomy extending above the stricture with repair by esophagogastrostomy and circumferential esophagofundopexy is the procedure of choice.

ILLUSTRATIVE CASES

R. B. was born in December, 1948, a full-term infant with normal development. His mother's history and pregnancy were not remarkable, and he weighed 6 pounds at birth. In June, 1949, he had a 2-week episode of hematemesis and vomiting; however, he ate with a good appetite. In July, 1949, he was admitted to the Pediatric Ward of the Denver General Hospital for recurrent intermittent hematemesis. He had failed to gain weight and vomited once daily. His gastrointestinal series at this time was considered to be normal. Following his hospitalization there was no blood in the vomitus, raised on an average of once daily for 1 month. In September, 1949, he again had recurrent hematemesis and daily spells of crying. Esophagoscopy at this time showed blood emerging from the cardia of the stomach, but no definite lesions were seen. Exploratory laparotomy was carried out with abdominal repair of a diaphragmatic hernia, appendectomy, and gastrotomy. The latter procedure demonstrated no gastric lesions. At this time he was considered to have a congenital short esophagus. The postoperative course was satisfactory.

Following this operation he had attacks of recurrent vomiting, the vomitus often containing blood and associated with some abdominal pain. He continued to eat well and to have a good appetite. In March, 1950, he was again hospitalized with severe recurrent hematemesis and anemia. A second exploratory laparotomy and gastrotomy at this time

demonstrated no site of bleeding. Fig. 10 shows an esophagram taken in February, 1952. In May, 1953, he was again admitted to the hospital with abdominal cramping pain, recurrent hematemesis, and vomiting. Esophagram at this time showed gastric mucosa in the lower thorax, but no gastric pouch or stenosis was noted. Esophagoscopy demonstrated an intrathoracic stomach with terminal esophagitis, and bleeding from the lower esophagus. On June 1, 1953, a left phrenic nerve crush was performed.

On June 5, 1953, a third laparotomy was carried out with repair of a recurrent diaphragmatic hernia. Postoperatively he had continuous nasogastric suction, and the course was uncomplicated. Between June, 1953, and March, 1954, he had occasional hematemesis and intermittent abdominal and substernal pain. His weight varied between 30 and 35 pounds without appreciable gain. In March, 1954, he again had severe hematemesis and abdominal pain which persisted after antacids and milk for the next 2 months.

Fig. 10.—R. B. (Case 1). February, 1952. Esophagogram demonstrating short esophagus and recurrent esophageal hiatal hernia.

Melena was present for 1 month. In May, 1954, a barium swallow showed a sliding esophageal hiatus hernia with gastric mucosa above the diaphragm and gastric retention. Esophagoscopy showed esophagitis of the lower third with bleeding at the cardia. A left transthoracic repair of the diaphragmatic hernia was attempted and incidental splenectomy and esophagorrhaphy were carried out. He made an uncomplicated recovery from this operation. In June, 1954, an esophagram showed no stricture at the esophagogastric junction. In February, 1955, he had a 1 week episode of abdominal pain, hematemesis, but

no melena. The hemoglobin at this time was 9 grams per cent, and stools were negative for blood. He weighed 35 pounds, and was placed on iron therapy. An esophagram demonstrated no hernia. Between February, 1955, and April, 1958, he had several upper respiratory infections with vomiting and some abdominal pain. His last hematemesis occurred in December, 1955. He slept with his head elevated and was taking multiple feedings. His weight had increased to 45 pounds. In April, 1958, he complained of marked tiredness in the morning and heartburn. His hemoglobin at this time was 9.8 grams per cent with 2.1 per cent reticulocytes. He was continued on iron therapy. In May, 1958, his stool guaiac was 4+ positive and hemoglobin was now 11.7 grams per cent. In July, 1958, an upper gastrointestinal x-ray series showed marked gastric reflux into the lower three quarters of the esophagus (Fig. 11), and a sliding esophageal hiatus hernia was seen.

Fig. 11.—R. B. (Case 1). July, 1958. Massive reflux of barium into the esophagus demonstrated by placing the patients in the Trendelenburg position following a barium meal.

There was some pylorospasm present. Esophagoscopy demonstrated an esophageal ulcer at 27 cm. with fresh bleeding and marked esophagitis. Gross incompetence of the cardia was present. X-ray studies in August, 1958, showed the magenblase above the left diaphragm (Fig. 12).

In September, 1958, left thoracotomy was performed. At operation 8 cm. of stomach were present in the posterior mediastinum. The lower third of the esophagus was markedly thickened, edematous, and attached to the subpleural connective tissue by rather dense, friable adhesions. However, since preoperative esophagoscopy had demonstrated that the esophagus could be easily dilated to full caliber, it was decided to perform circumferential esophagofundopexy without resecting that portion of the lower third of the esophagus

which was obviously the site of esophagitis. The gastric pouch was separated from the diaphragmatic orifice to which it was attached by firm adhesions so that the stomach could be more easily drawn up into the thorax. A line of sutures of interrupted 3-0 silk were placed between the left border of the esophagus and the right border of the gastric fundus for a distance of 6 cm. Then the posterior and anterior surfaces of the fundus of the stomach were wrapped around the distal third of the esophagus and approximated snugly with two layers of interrupted 3-0 silk sutures. This layer of sutures was continued to a point 2 cm. below the inosculation of the esophagus into the gastric cardiac so as to protect the squamous epithelium of the cardia from the acid-pepsin gastric juice. The stomach was then anchored to the esophageal-diaphragmatic orifice with interrupted 3-0 silk sutures. Following the placement of two intercostal chest tubes, the chest wall was closed in layers. An indwelling nasogastric tube had been placed in the stomach preoperatively, and this was allowed to remain until the third postoperative day. He made an uncomplicated recovery and has had no attacks of hematemesis since that time. Postoperative chest film shows a gas bubble posterior to the heart. His weight was 45 pounds at discharge.

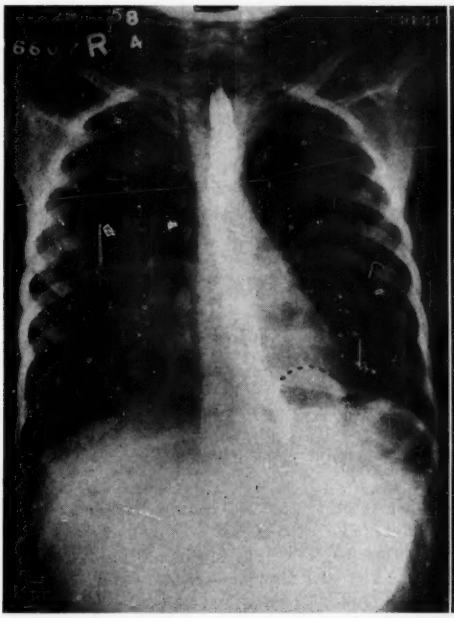

Fig. 12.

Fig. 13.

Fig. 12.—R. B. (Case 1). Aug. 29, 1958. The magenblase is well seen above the left diaphragm, indicating an esophageal hiatal hernia.

Fig. 13.—R. B. (Case 1). Dec. 1, 1958. Postoperative upper gastrointestinal series shows the esophagus and stomach during the act of swallowing a large bolus of barium. The extent of the Witzel tunnel is demonstrated, and the barium is seen to enter the stomach readily.

In December, 1958, an esophagram demonstrated no reflux with the patient in a marked Trendelenburg position. The cardia was seen to fill above the diaphragm, and there was no dilatation of the esophagus (Fig. 13). In January, 1959, esophagoscopy demonstrated no esophagitis and no reflux in the supine or Trendelenburg position. There was good closing of the terminal esophagus. At this time the hemoglobin was 15.4 grams per cent, and he weighed 48 pounds. In March, 1959, an esophagram demonstrated no esophageal reflux or stricture.

Comment: This boy had been subjected to four attempts to repair what is obviously an esophageal hiatal hernia of the congenital short esophagus type with recurrent ulcerative esophagitis. In spite of repeated efforts to place the stomach below the diaphragm, the shortening of the esophagus was such that this maneuver could not be accomplished, and the hernia promptly recurred. He had suffered from recurrent ulcerative esophagitis with hematemesis since his nursling days and had received only moderate amelioration by any of the procedures which had been carried out. Recognition of the fact that esophagitis was the serious component of this patient's lesions, and dispossessing ourselves of the preoccupation to produce anatomic restoration of the concerned viscera, we felt that this boy presented an ideal indication for trial of our investigative circumferential esophagofundopexy. His prompt and gratifying response to this procedure gives us encouragement, although repeated long-term observation will be necessary to establish the worth of this undertaking.

M. K., a 62-year-old white waitress, had been seen in the Medical Outpatient Department from May of 1956 to January of 1957. She noted abdominal epigastric, and sometimes sharp substernal aching pains which had begun about 7 years previously, with recurrences of this pain at frequent intervals since that time. The pain was aggravated by lifting, stooping, straining on exertion, and by the ingestion of milk. The pain was lessened or decreased by the use of phenobarbital, retirement to bed with a hot water bottle, or discontinuance of activity. Pain was not related to meals; walking did not accentuate it. There was no associated nausea, vomiting, melena, or hematemesis. There was occasional radiation of pain into the back. Her weight had remained stable at 100 pounds.

Back pain associated with stiffness and soreness had been diagnosed as arthritis with response to appropriate therapy. She had had an appendectomy in 1929. A symptomatic right renal stone had been diagnosed in 1950 with abdominal ureterolithectomy in 1952 without relief of all symptoms. She had no frank pyelitis, but urinary frequency. Two gall bladder series taken 1 year prior to her first visit to Denver General Hospital were reported normal. Examination disclosed blood pressure to be 124/70 mm. Hg, pulse was 76 and regular, respirations were 16, and she weighed 102 pounds. Two healed scars were present in the right lower quadrant, and there was mild right lower quadrant tenderness which was superficial.

Laboratory examinations showed that hemoglobin was 14.9 grams per cent, total leukocyte count was 9,800 with a normal differential. Urinalysis was negative. Blood urea nitrogen was 8 mg. per cent; uric acid, 5.5 mg. per cent; fasting blood sugar, 107 mg. per cent. An electrocardiogram was negative. The gall bladder series showed a normal functioning gall bladder without stones. An upper gastrointestinal series showed a moderate-sized hiatus hernia with one fourth of the stomach above the diaphragm. The cervical and lumbar spine films showed osteoporosis with hypertrophic changes. An intravenous and retrograde pyelogram showed kinking of the right proximal ureter due to previous surgery without obstruction to the pelvis of the kidney and no dilatation of the ureter.

The patient was treated following her first visit with phenobarbital and belladonna. There was no relief of pain. She was seen over the next 5 months in several medical clinics with the same problem of abdominal pain. She was placed for a time on Peritrate and nitroglycerine without relief of pain.

In January, 1957, she was referred to the Department of Surgery and admitted to hospital. On Jan. 4, 1957, esophagoscopy showed redundant distal esophageal folds with a red, granular, friable bleeding mucosa without gross ulceration. No stricture was noted.

On Feb. 11, 1957, this patient had repair of a sliding esophageal hiatus hernia by the left thoracic approach. Over half of the stomach was present in the posterior mediastinum and, despite considerable mobilization of the stomach and esophagus, the stomach could not be reduced below the diaphragm. Despite the fact that there was no concentric narrowing of the distal esophagus, it was believed that the protracted bouts of peptic esophagitis which she had experienced had produced longitudinal shortening of the esophagus. Consequently, circumferential esophagofundopexy was carried out in the manner described above. Following this, two thirds of the stomach was reduced below the diaphragm leaving one third, including the esophagogastric junction, in the posterior mediastinum. The postoperative course was uneventful, and the patient was discharged on the eleventh postoperative day. Subsequent clinic visits have shown her to be in good health with no further abdominal symptoms, and no evidence of peptic esophagitis. This clinical impression has been verified by repeat esophagoscopic examinations.

Comment: This patient had developed incompetence of the gastric cardia secondary to the long-standing esophageal hiatal hernia. The regurgitative peptic esophagitis which then developed had resulted in damage only to the esophageal mucosa insofar as concentric narrowing is concerned. However, it was obvious at the time of operation that there was shortening of the esophagus in a longitudinal direction. In those cases in which the esophagus can be dilated to full and sufficient caliber, we have been performing circumferential esophagofundopexy to control the peptic esophagitis and have not resected the distal esophagus. Resection of the lower third of the esophagus has been reserved for deep strictures which could not be dilated.

J. W., a 76-year-old white male old age pensioner, entered Denver General Hospital on May 10, 1958, with the complaint of dysphagia for 4 years and regurgitation of food for 1 month. He had developed progressive increase in dysphagia during the preceding month and had lost 20 pounds in weight. During the month preceding admission he had been unable to swallow any solids, and upon admission he was unable to take even water.

Examination disclosed a poorly nourished, moderately well-developed white man who appeared chronically ill. Blood pressure was 110/75 mm. Hg, pulse 84, respirations 24. Examination of the chest showed some increase in anteroposterior diameter. The lungs were hyperresonant and the breath sounds were decreased in intensity. A systolic murmur was present over the apex. The remainder of the physical examination was not remarkable.

The urinalysis was negative on admission. Examination of the blood demonstrated a hemoglobin of 11 grams; white blood count was 12,400 with a normal differential. Total blood proteins and the albumin-globulin ratio were normal. The serology was negative. An electrocardiogram showed normal sinus rhythm with some enlargement of the left ventricle. A roentgenogram of the esophagus showed a marked obstruction in the lower third at a point 8 cm. proximal to the cardia.

Esophagoscopy demonstrated an obstruction in the lower third of the esophagus. The obstruction consisted of very firm, yellow, dense tissue which grossly appeared to be granulation tissue. This impression was confirmed by microscopic examination.

On May 19, 1958, a left thoracotomy was performed through the bed of the eighth rib. The lung showed chronic fibrosis and emphysema with a few scattered subpleural blebs. The posterior mediastinum was opened and the lower third of the esophagus was found to be densely adherent to the surrounding cellular tissue and lymph nodes. Approximately 8 cm. of stomach was present above the diaphragm in the posterior mediastinum. The esophagus was freed from its bed with difficulty and there was considerable bleeding. The lesion extended over an area of 5 cm. and the fibrosis involved the entire wall of the esophagus. The diaphragm was opened from the esophageal hiatus outward

through the central tendon, and the stomach was mobilized along the greater and lesser curvatures, ligating the left gastric and left gastroepiploic vessels, but leaving the spleen intact. The esophagus was cut across at the cardia approximately 6 cm. below the esophagogastric junction, and the cardia was closed. The stomach was sufficiently mobilized so that it could be easily drawn up into the left hemithorax. The esophagus was transected 2 cm. above the superior edge of the lesion and this segment was removed. Esophagogastrostomy was carried out, utilizing an inner layer of chromic 3-0 catgut and an outer layer of interrupted 3-0 silk sutures; the stoma of the esophagogastrostomy was placed well down on the lesser curvature, almost at the antrum of the stomach. Following completion of the esophagogastrostomy, the lesser curvature of the stomach was approximated to the left border of the esophagus with interrupted 3-0 silk sutures. Following this, circumferential esophagofundopexy was carried out in the manner described above. The imbricating sutures were extended along the lesser curvature to a point well below the stoma of the esophagogastrostomy. Following completion of this procedure the stomach was anchored to the hiatus of the diaphragm with interrupted 3-0 silk sutures, and the chest was closed in layers, following the placement of two No. 45 Pezzer catheters. The patient's postoperative course was unremarkable.

Follow-up roentgenographic and esophagographic examinations have shown the absence of esophagitis and the patient has gained 45 pounds in weight.

Comment: This case illustrates the devastating effect of long-standing, neglected peptic esophagitis associated with the sliding type of esophageal hiatus hernia. Here it was impossible by dilatation to achieve any lumen in the distal third of the esophagus because of the great density and extent of the stricture. In such a case, in order to re-establish a lumen, resection of the stricture must be carried out. In this particular case, submucosal resection of the stricture according to the technique of Allison was considered, but it was impossible to develop a reasonable cleavage plain preserving the muscularis of the esophagus. For this reason resection of the strictured area was carried out with the reconstructive procedure as stated above.

SUMMARY

1. Experimental investigations are reported from which the procedure of circumferential esophagofundopexy has been developed.

2. The application of this procedure to cases of brachyesophagus and peptic ulceration of the lower third of the esophagus are discussed and several illustrative cases are presented.

REFERENCES

1. Quincke, H.: Ulcus oesophagi ex digestione, Deutsche Arch. f. Klin. Med. **24:** 72, 1879.
2. Briggs, P. J., Dick, R. C. S., and Hurst, A.: Simple Ulcer of Oesophagus and Short Esophagus, Proc. Roy. Soc. Med. **32:** 1423, 1939.
3. Barrett, N. R.: Hiatus Hernia—A Review of Some Controversial Points, Brit. J. Surg. **42:** 231, 1954.
4. Valdoni, P.: Traitement radical des stenoses oesophagiennes; La prevention de l'oesophagite peptique post-operatoire, Presse méd. **59:** 1216, 1951.
5. Watkins, D. H., Prevedel, A., and Harper, F. R.: A Method of Preventing Peptic Esophagitis Following Esophagogastrostomy, J. THORACIC SURG. **28:** 367, 1954.
6. Franke, H.: Zur Frage der Vermeidung einer Reflux oesophagitis nach Kardiaresektion, Arch. f. klin. Chir. **287:** 407, 1957.
7. Adler, R. H., Firme, C. N., and Lanigan, J. M.: A Valve Mechanism to Prevent Gastroesophageal Reflux and Esophagitis, Surgery **44:** 63, 1958.

Discussion

DR. WILLIAM E. NEVILLE, Cleveland, Ohio.—At the moment, having spent 3 days here in Los Angeles, I feel more competent to discuss the symptoms of esophagitis rather than the surgical management of it.

Two years ago, before this Association, my associate, George Clowes, and I had the opportunity to present our results in colon replacement of the esophagus. The satisfactory results in patients operated on for carcinoma led us to extend this operative procedure to other conditions involving the esophagus.

In order to evaluate whether it would be feasible to replace the lower end of the esophagus with the colon, we took the problem to the laboratory.

[Slide] This is an autopsy specimen taken from a dog operated upon and sacrificed at the age of 9 months. This is the esophagus; this is the transplanted colon segment; and this is the stomach and, as you can all see, there is no evidence of esophagitis, colitis, or gastritis.

At that time we thought this was a pretty severe test for the efficacy of this procedure, due to the fact that all of these dogs had marked regurgitation.

[Slide] Commensurate with this, we began seeing patients who died as a result of progression of their disease up to 2 to 3 years following the operation for the resection of carcinoma. This shows the esophagus which, as you can all see, is normal in appearance, and this is the transplanted colon segment.

[Slide] This is another patient who succumbed to the ravages of his disease. This is a microscopic section, as you can all see, right at the esophagocolon anastomosis. This shows normal-appearing mucosa at the level of the esophagus, and this is, of course, the colon transplant with no evidence of ulceration.

[Slide] This patient was operated upon, having come in with a severe esophagitis with a recurrent hiatal hernia. Repeated dilatations failed to alleviate the symptoms, and for this reason we resected the esophagus and interpolated a segment of transverse colon between the upper end of the esophagus and the stomach. This man has been completely restored to normalcy as far as his esophagus is concerned.

[Slide] This last slide shows a lesion in the lower end of the esophagus with stenosis for which resection was done and the colon interpolated between the esophagus and the stomach.

We have only operated upon six patients to date for severe stenosing esophagitis, but it does seem to be a reasonable approach to the problem in the severe disabling form of this disease.

DR. J. GORDON SCANNELL, Boston, Mass.—I have enjoyed Dr. Watkins' description of this most ingenious procedure very much, but I rather hope that in closing he will tell us how many cases, and how long these have held up.

DR. CAMERON HAIGHT, Ann Arbor, Mich.—The procedure which Dr. Watkins has illustrated has, I believe, a great deal to offer. I am very much intrigued by it and I would particularly like to ask him if he has used it in conjunction with any colon transplant operations, because it would seem, a priori, that it might have a decided advantage in such cases.

I would like to mention one physiologic disadvantage of a right colon transplant. It offers no protection against regurgitation from the stomach into the transplanted colon. This can be readily demonstrated by x-ray examination. When barium has been introduced through the gastrostomy tube postoperatively with the patient in the upright position, the barium remains largely in the stomach. However, when the patient is placed in the supine position, there is very extensive and prompt regurgitation of the barium into the transplant. This is undesirable physiologically because it allows retention of feedings in the transplant when the patient is in the supine position and asleep. It also allows air to come up freely into the transplant.

Fortunately, however, with the colon there does not seem to have been acid irritation and ulceration of the transplant, as observed by examination of the stomach and adjacent colon with a cystoscope introduced through the gastrostomy.

We have used a right colon transplant in 3 adults who have had strictures of the lower esophagus either from peptic esophagitis or from caustic causes. Our reason for the choice of the colon for reconstruction of the esophagus was that all patients previously had extensive gastric resections because of the accompanying pyloric lesions. It was not believed possible to resect the extensive strictures of the esophagus in these cases and to bring the remaining small portion of the stomach high enough into the chest to perform an esophagogastrostomy. A right colon transplant would appear to be particularly useful in such patients who have previously had a very extensive subtotal gastric resection in conjunction with peptic esophagitis.

DR. F. HENRY ELLIS, JR., Rochester, Minn.—I would like to comment primarily on the theoretical basis of Dr. Watkins' interesting surgical procedure. I inferred from his presentation that he believes the esophagogastric angle is chiefly responsible for preventing regurgitation under normal conditions. Considerable controversy exists regarding the mechanism that prevents gastroesophageal regurgitation, but there is increasing evidence that the esophagogastric angle not only may not exist at all but, if it does, that it does not contribute to the prevention of reflux.

Physiologic studies give increasing evidence of the fact that there is a physiologic, if not an anatomic, esophageal sphincter that, together with some other factor such as a valve-flap mechanism at the cardia, may be of chief importance in preventing reflux.

Experimentally, Dr. Meiss in our laboratories has shown that the gastroesophageal angle can be completely obliterated provided the esophagogastric junction is not disturbed. Thus, our results were opposite to those Dr. Watkins has described in carrying out experimental fundusectomy. I would like to ask him more about the technique of his fundusectomy. I wonder if, in his experiments, he perhaps interfered in some way with the integrity of the esophagogastric junction in the process of excising the gastric fundus.

DR. WATKINS (Closing).—I would also like to emphasize that we are talking about a procedure which is applicable to a very small group of patients who represent the end and devastating result of peptic esophagitis which should have been treated much earlier by the methods which Dr. Cross emphasized previously.

I don't think from our experiments, whether they are animal or whether they are simple observations on postmortem specimens, that there is any doubt but that the angle furnishes some type of augmentation. Whether this is a mucosal flap or some other structure I can't say. I think it is a mucosal flap. This is augmented by having an acute angle between the esophagus and the stomach. It is easy to demonstrate this by applying water pressure through the pylorus into the stomach with the fundus of the stomach isolated from the body by a clamp or by augmenting it by excising the left leaf of the diaphragm.

The fundusectomies which were carried out here experimentally dividing the sling muscle as best we could do it technically.

We have not used colon transplants, in answer to Dr. Haight's question, in combination with this procedure. We have used left colon transplants to replace the esophagus, placing it substernally following total esophagectomy according to the technique of Orsoni.

I have used colon in this place because I have always thought the adequate mucous production by the colon offered more adequate protection against acid peptic digestion than did jejunum.

The observations of Dr. Ellis regarding a functional sphincter in the distal esophagus are most interesting. Dr. Lerch, who has been a member of this Society for many years, demonstrated to his satisfaction but not to that of all anatomists that such a structure does actually exist. We have not been able to demonstrate it.

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